



# Clinical Applications *of* Bronchology

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Dedicated  
to  
CHEVALIER JACKSON  
Father of Modern Bronchoesophagology



# Foreword

Dr Kassay's book *Clinical Applications of Bronchology* is unique in the English language. It is a mature work based on a comprehensive personal clinical experience and on a profound knowledge of the field of diseases of the bronchi and the techniques of their study and treatment.

The first two chapters of the book present a concise review of applied anatomy and physiology of the bronchi taking into consideration the modern concept of the bronchopulmonary segment and describing the various valvular mechanisms which must be understood in order to comprehend and interpret physical signs.

The chapter on roentgenology deals with not only the roentgenographic appearances but especially with fluoroscopic appearances. Elaborate and unusual consideration is given to the need to recognize the Holzknacht sign and its significance in the diagnosis of bronchial obstruction. The x-ray appearance of the pulmonary units particularly of the segments and subsegments of the lungs is beautifully presented.

Instruments techniques and indications for bronchoscopic and bronchographic examination are discussed in a practical way particular attention being given to the author's own technique of aimed bronchography.

An entire chapter is devoted to diseases with valvular respiratory mechanisms and another to infectious and allergic bronchitis. Other conditions which are presented especially from the bronchoscopic and bronchologic viewpoint are pneumonia, pulmonary abscess, bronchiectasis, pulmonary tuberculosis and mycosis and bronchial tumors. Among bronchial tumors particular consideration is given to bronchial adenoma.

Foreign bodies are discussed in a separate chapter and as he does with other subjects the author illustrates his points with typical cases from his own clinical experiences.

Differential diagnosis is the subject of the final chapter with particular reference to the role of bronchoscopy and bronchography.

The Selected References includes the most recent contributions to the literature by both American and foreign authors and is exceptionally well prepared. The Index is comprehensive and its cross references make it most useful.

*Clinical Application of Bronchology* constitutes a real addition to the literature of bronchology and will be read with interest and profit by practitioners of internal medicine, pediatricians, roentgenologists, and surgeons, as well as by bronchologists

CHEVALIER L. JACKSON, M D

# Preface

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In the sixty years since its development the bronchoscope has come to be regarded not only as an instrument for the removal of foreign bodies from the bronchi and their adjacent organs but also as an invaluable diagnostic and therapeutic instrument. As a result of the possibilities seen for clinical applications of the bronchoscope a new and necessary medical specialty came into being—that of bronchology, the study and treatment of the bronchial tree.

With the improvement of the bronchoscope and with the recognition of its versatility the use of the instrument in the diagnosis and treatment of diseases of the lungs and their associated structures is of increasing interest and importance. Bronchoscopic examination in conjunction with the usual physical, x-ray, and laboratory examinations now enables the physician—the general practitioner, pediatrician, internist, or surgeon—to diagnose diseases or dysfunctions of the entire respiratory tract or a minute part of it with greater accuracy and with less discomfort for the patient.

Because this book is written for general practitioners and other physicians who are interested in bronchology and who may have occasion to use the bronchoscope in their practice, basic techniques for its use, premedication, medication, and anesthetics, positioning of the patient, and other practical information are described in detail.

Of course not every condition which might indicate bronchoscopic procedures is included in this book. However, those most frequently encountered are discussed in detail, and the general principles are applicable in the investigation and treatment of most conditions.

Many physicians still have the notion that the bronchologist's function is merely to provide a description of the color and condition of the mucosa or the width of the bronchial lumens. This notion is basically incorrect for the bronchologist, by his training and experience, can also discern and interpret the finer nuances of abnormalities, such as small deformities, accordion-like motion, a lateral shift of the tracheal carina, dullness of the bronchial spurs, type, number, and size of spots of secretion, or degrees of diminished bronchial movements, and therefore he is being called upon more often to evaluate and to aid in the solution of problems related to diseases of the lungs.

It is the hope of the author that the material presented here will be used by the physician as an up-to-date guide for bronchoscopic proce-



dures and that it will show how the experience and the techniques of the bronchologist can be of value in discerning and diagnosing diseases of the lungs

*It is now my pleasure to acknowledge and thank some of the people who gave me such earnest cooperation in my clinical work in Budapest. Among them are Jozsef Balo, pathologist, Ferenc Kováts, Sr., and Ferenc Kováts Jr., pulmonologists, Oszkár Gorgényi, Gottche and Géza Petenyi, pediatricians, Mihály Erdélyi, roentgenologist, Imre Kerenyi, András Bikfalvi, and Attila Balás, chest surgeons. My immediate talented and devoted assistants were Ilona Szekács, Zoltán Selymes, Zoltán Lábás, and György Mihók, otolaryngologists, and Mária Fazekas, head nurse, and Anna Sándor, operating room nurse.*

To Dorothy D. Robinson, who made many of the drawings for the book, I wish to express my sincere appreciation for her excellent work.

I am especially indebted to the following authors for the privilege of reproducing figures and tables from their works: Maurice T. C. Bariéty, Russel Brock, Edwin N. Broyles, Julius E. Comroe, Jr., Nolan L. Kaltreider, Ferenc Kováts, Jr., Arthur Proetz, Johannes A. G. Rhodin, and Ernst Stutz.

Finally, I wish to express my sincere gratitude to Dr. Chevalier L. Jackson for his interest and efforts on my behalf in the publishing of this book, for providing helpful suggestions, and for honoring it with a foreword, to Dr. Charles M. Norris for his guidance in the many problems one faces in translating a technical work, and to Dr. Ernest L. McKenna for the long hours he spent in correcting the English text.

DEZSO KASSAY, M.D.

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# Anatomy

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## THE BRONCHIAL TREE

The tracheobronchial system together with the primary lobuli resembles a leafy tree. The *trachea* represents the trunk of the tree, the *bronchi* are the branches and the *primary lobuli* the leaves. The term "lung tree" is descriptive not only of the shape of the tracheobronchial system but also of its functions. In the "lung tree" as in real trees the branches—the bronchi up to and including the terminal bronchioli—do not take part in respiration. The respiratory organs of a tree are its leaves and similarly the breathing parts of the lung are the respiratory bronchioli, the alveolar ducts and the alveoli. The alveolar ducts and the alveoli comprise the primary lobuli (Fig 1) where as in leaves oxygen and carbon dioxide are exchanged. Two or three primary lobuli comprise an acinus. The term "lung tree" could be used to refer to the whole aerated interior of the lung and bronchial tree to the larger branchings. However since the expression *bronchial tree* is so deeply rooted in routine medical work we shall use it in this book. The term "lung tree" will be used when referring to some processes in which not only the bronchial tree but the alveoli are included.

In this monograph upper and lower airways will be mentioned in several instances. The term *upper airway* means the air tract above the glottis and the term *lower airway* means the air tract below.

A physician should be familiar with the segmental branchings of the bronchial tree. Without this knowledge he cannot grasp the meaning of up to date records of a pneumologist, roentgenologist or bronchologist. He cannot analyze bronchograms and perhaps not even a simple x-ray film. Thus, he may be left decades behind modern pneumology.

Many older physicians did not learn the modern classification of the bronchi. Two decades ago the medical schools were teaching the system of the primary, secondary, tertiary, etc. branchings of the bronchi as well as the "eparterial" and "hyparterial" bronchi. Today the latter system is seldom used, the latter one never. According to the *Arbeitskreis*

the primary bronchus is the main bronchus the secondary the lobar bronchus the tertiary the segmental bronchus and the quaternary one the subsegmental bronchus. In general modern classifications deal with the segmental bronchus but in some investigations the subsegmental bronchus has been described. The segmental bronchus is a branch of the lobar bronchus the subsegmental bronchus is a branch of the segmental bronchus.

The committee of the Fourth International Congress of Otolaryngology (London 1949) discussed the question of classification and nomenclature

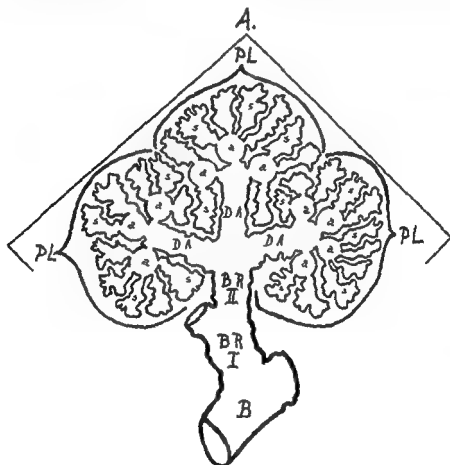


FIG. 1. Branching of a secondary lobe. A. acinus. PL. primary lobule. B. bronchiole (terminal bronchiole). BR I. respiratory bronchiole I. BR II. respiratory bronchiole II (alveolar bronchiole). DA. alveolar duct. a. alveolus. (From W. S. Miller, *The Lung*, 1950. Courtesy of Charles C. Thomas, Publisher, Springfield, Illinois.)

of the bronchial tree and came to an agreement<sup>22</sup> The international nomenclature is however only a modification of and in some aspects is not as correct as the Jackson Huber system<sup>23</sup> The new nomenclature was criticized by Boyden<sup>24</sup> and the author<sup>25</sup> Because of the international agreement the London nomenclature is used in this text however we add two subsegmental bronchi more important than the two (1 and 2 of the left side) that appeared in the new nomenclature (see the diagram later in this chapter) We mention too the subapical segmental bronchus of the lower lobes which should also be included in the nomenclature

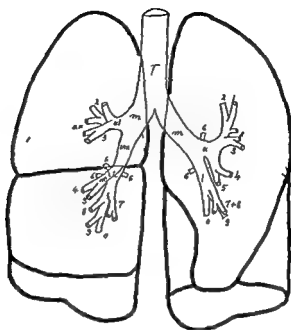
### Description

**The Trachea** The *trachea* directed slightly backward and to the right divides into *right* and *left main bronchi* (Fig. 2) The spur between the two main bronchi is called *tracheal carina* or *carina tracheae* It is a sharply ending partition between the airways of the two lungs and a very important landmark for the bronchologist during bronchoscopy

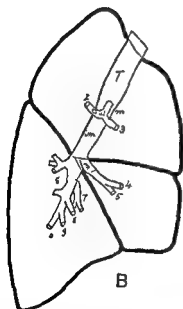
**Right Lung** The right main bronchus is about 2 cm long and is wider than the left one It lies almost in the axis of the trachea Running downward it deviates slightly from the vertical laterally and backward The right main bronchus divides into two branches the *upper lobe bronchus* and the *stem bronchus* (*bronchus intermedius*) The latter follows the direction of the main bronchus Its lumen is narrower than that of the left main bronchus It divides into *middle* and *lower lobe bronchi*

The *right upper lobe bronchus* is directed laterally and runs almost horizontally at a right angle to the main bronchus In approximately 70 per cent of the cases it divides into three branches in 20 per cent into two branches and in 10 per cent into four branches (candelabra position) The three segmental branches are the *apical posterior* and *anterior* In the majority of cases there are two axillary subsegmental branches One originates from the posterior bronchus the other from the anterior In most cases of bifurcation the upper posterior bronchus divides into the apical and posterior branches the lower anterior bronchus into the anterior and an axillary branch In the "candelabra" position the four bronchi are represented by the above three segmental bronchi plus an axillary branch which in this case is considered a segmental bronchus<sup>26 27 28</sup>

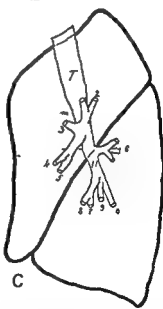
The *apical segmental bronchus* supplies the apex of the lung the *posterior bronchus* supplies the posterior portion of the upper lobe and sometimes the posterior portion of the apex as well The *anterior segmental bronchus* supplies branches for the anterior portion of the upper lobe that lies under the pectoral muscles The *axillary subsegmental bronchi* as the name implies supply the axillary portion of the upper



A



B



C

FIG 2 The bronchial tree up to the segmental and subsegmental branching. T trachea m main bronchus in stem bronchus (1 incl. intermediate) l upper lobe bronchus ml middle lobe bronchus ll lower lobe bronchus l lingular bronchus ax axillary subsegmental bronchus. The numbers refer to the number of each segment (see page 9) A Anterior view B C Lateral view

lobes. The subsegment supplied by this branch or branches is important because atelectasis, abscesses, epituberculous changes, etc., frequently appear in this region.

The *middle lobe bronchus* originates on the anterior wall of the stem bronchus (*bronchus intermedius*) 1 to 3 cm below the upper lobe bronchus. It runs forward, downward, and slightly laterally, sometimes forming an S curve. Its two branches are the *lateral* and the *medial segmental bronchi*. The lateral bronchus runs laterally and forward and supplies the portion of the middle lobe that lies under the mamma. The medial bronchus is directed forward and slightly downward, supplying the medial portion of the middle lobe that is in contact with the heart and sternum.

The *right lower lobe bronchus* follows the direction of the main bronchus and stem bronchus. Whereas the bronchi of the upper and middle lobes are considered as *lobar bronchi* up to their first branching, the lower lobe bronchi are referred to as *lower lobar bronchi* as far as the terminal basal branching, even though two or three segmental branches have originated before this point. Thus the lower lobar bronchus spreads its segmental branches not all from the same point as do the upper and middle lobar bronchi (dichotomy), but segmental bronchi leave the lobar bronchus one after another (monopodium).

The first branch of the lower lobe bronchus is the *apical segmental bronchus*. This branch originates on the posterior wall of the lower lobe bronchus just opposite or slightly below the middle lobe bronchus. It divides into three branches: superior, medial, and lateral. In an examination of a bronchogram, knowledge of these three bronchi is necessary. The apical segmental bronchus supplies the upper portion of the lower lobe.

The second branch of the lower lobe bronchus is the *medial basal segmental bronchus*, originating from the medial aspect of the lobar bronchus midway between the apical and anterior basal branches. This branch supplies the anterior medial basal portion of the lower lobe in contact with the diaphragm, heart, and the lower mediastinum.

The next branch is the *anterior basal segmental bronchus*, supplying the anterior lateral basal portion of the lower lobe. However, in about 50 per cent of the cases, a large bronchus (the subapical), which should also be mentioned, originates opposite to and a little above the origin of the anterior basal branch. It supplies the lung tissue below the apical segment.

Below the anterior basal bronchus is the common trunk of the *lateral* and the *posterior basal segmental bronchi*. The common trunk runs but a short distance before it divides into these two basal branches. The lateral basal bronchus supplies branches to the lateral posterior basal



portion of the lower lobe. The posterior basal bronchus extends to the lowest portion of the lung and therefore is called the *terminal bronchus* by many writers.<sup>19</sup>

**Left Lung.** The left main bronchus is 4.5 to 5 cm long and divides into the *upper and lower lobe bronchi*. The upper lobe bronchus runs forward and laterally and after a distance of from 1 to 1.5 cm divides again into *upper and lower divisions*.

The upper division turns sharply upward in a J curve and supplies the upper part of the left upper lobe which corresponds to the right upper lobe.

The lower division is generally called the *lingular bronchus* and the area supplied by it the *lingula*. Originally the term *lingula* was applied only to the lower (tongue shaped) portion of the left upper lobe located in the anterior phrenicocostal sinus; recently the name *lingula* is applied to the lower division of the upper lobe that corresponds to the right middle lobe.

The author has named the upper portion of the left upper lobe which is supplied by the upper division bronchus the "truncated lobe" and its bronchus the "truncated lobar bronchus."<sup>20, 21</sup> The term "truncated lobe" is applied to this part of the lung because *upper division* refers only to the bronchus and not to the unit of the lung. In creating this term the left upper lobe was separated in theory from the lingula. The use of the term *truncated lobe* implies that although this portion of the lung is accepted in the segmental classification and in clinical work as a lobe in reality and anatomically it is only a portion of the upper lobe; thus it is truncated. The new term seems correctly to solve the controversy between pure anatomy and both accepted classification and clinical routine. In the following description the *truncated lobar bronchus* and the *lingular bronchus* are considered as lobar bronchi homologous to the right upper and middle lobar bronchi.

The truncated lobar bronchus (upper division) divides into two branches the *apical* and *anterior segmental bronchi*. The apical segmental bronchus divides into the *apical* and *posterior subsegmental bronchi*.

The third segmental bronchus is the *anterior* one which on the left is larger and in the apical direction supplies a wider area than the right one.

The *axillary subsegmental bronchi* in this lobe show a wide variation but generally two axillary branches appear: one from the apical and the other from the anterior segmental bronchus.

The *lingular bronchus* divides into *superior and inferior segmental bronchi*. The lingular superior one supplies the upper portion and the lingular inferior one the lower portion of the lingula. The inferior branch

cludes the tongue shaped portion extending into the anterior phrenico costal sinus

The first branch of the left lower lobe, as in the right lower lobe, is the *apical segmental bronchus*

The *anterior basal segmental bronchus* is generally the second one originating from the lower lobar bronchus on the left. The common trunk divides further into the *lateral* and *posterior basal segmental bronchi*. On the left the basal branches arise more closely to each other, the division of these left basal branches frequently is seen at the same level.

The bronchial tree of the left lower lobe differs from that of the right lower lobe but their branchings are essentially comparable. The main difference lies in the fact that on the left in most cases the *medial basal branch* arises not from the lobar bronchus but from the anterior basal bronchus representing its first large medial branching. Nevertheless in 8 per cent of the cases the left resembles the right in that the medial basal branch originates from the lobar bronchus at a similar location. One other difference is that the *subapical branch* is not found so often on the left (in 8 per cent of the cases) as on the right (in 50 per cent).

### Nomenclature

The following is a diagram of the nomenclature used in this book, consisting of the London nomenclature<sup>30</sup> and the additional bronchi given in parentheses

RIGHT LUNG		LEFT LUNG	
Lobes	Segments	Lobes	Segments
Upper	<ul style="list-style-type: none"> <li>1 Apical</li> <li>2 Posterior</li> <li>3 Anterior (Axiillary)</li> </ul>	Upper	<ul style="list-style-type: none"> <li>1 2 Apical posterior</li> <li>3 Anterior (Axiillary)</li> </ul>
Middle	<ul style="list-style-type: none"> <li>4 Lateral</li> <li>5 Medial</li> </ul>	Lingula	<ul style="list-style-type: none"> <li>4 Superior</li> <li>5 Inferior</li> </ul>
Lower	<ul style="list-style-type: none"> <li>6 Apical (Subapical)</li> <li>7 Medial basal or cardiac</li> <li>8 Anterior basal</li> <li>9 Lateral basal</li> <li>10 Posterior basal</li> </ul>	Lower	<ul style="list-style-type: none"> <li>6 Apical (Subapical)</li> <li>7 8 Anterior (Medial anterior) basal</li> <li>9 Lateral basal</li> <li>10 Posterior basal</li> </ul>

## STRUCTURE OF THE BRONCHIAL WALL

To understand the diseases of the bronchi we must know the structure of the bronchial wall, which can be described as consisting of the following four main parts

- 1 The *trachea*
- 2 The *primary cartilaginous bronchi* These are the bronchi between the trachea and the origins of the apical segmental bronchi of the lower lobes. Their structure is similar to that of the trachea, their lumens are kept open by C-shaped cartilages, the posterior wall, corresponding to the open portion of the C, is membranous (the *paries membranaceus*). The caliber alterations of the trachea and of these bronchi are made possible by this membrane.
- 3 The *secondary cartilaginous bronchi* Small plaques or lamellae of cartilage are imbedded in the soft tissue of the wall of these bronchi. These cartilages never form a continuous circle, thus the lumens of these bronchi are able to widen and narrow.
- 4 The *intralobular bronchi* These are less than 1 mm in diameter and contain no cartilage.

The walls of the bronchi are composed of four layers.

- 1 The *mucosa*, which in turn has four layers
  - a The *epithelium*
  - b The *tunica mucosae*
  - c The inner elastic layer
  - d The muscular layers
- 2 The *submucosa*
- 3 The *fibrocartilaginous layer*
- 4 The *peribronchium*

Besides the cartilages the most important components of the bronchial wall are the epithelium, the pulmonary tonsil, the bronchial glands, the muscles, the elastic fibers, and the diverticula.

- 1 The *epithelium* The epithelium consists of single-layered ciliated cells. Among the epithelial cells are also active goblet cells. The length of a cilium is equal to the diameter of an erythrocyte (Fig. 4C). Ultrastructure of the ciliated cell is demonstrated in Figure 4D and E. The ciliary epithelium plays an important role in the physiology and pathology of the lungs.
- 2 The *pulmonary tonsil* The accumulation of the lymphocytes in the bronchial epithelial tissue may be so great and dense that it resembles the palatine tonsils. Collectively these structures are called *pulmonary*

tonsil by Hayek.<sup>29</sup> Characteristically the bronchial epithelium which covers these structures has no cilia.

- 3 The *bronchial glands*. These are mixed glands both serous and mucous. They are scattered rather thickly throughout the bronchial wall. Many of them are deeply imbedded and extend to the peribronchium beneath the cartilages. Their efferent ducts are extremely long and penetrate the several layers of the wall including the muscles. As a result diseases of the wall and pathologic action of the muscles may easily obstruct the efferent ducts and cause accumulation of the secretion in the glands. This unusual penetration of the glands is an important factor in chronic and deforming bronchitis and bronchiectasis and in the pathology of the "iceberg type" of bronchial adenoma. In the intralobular bronchi which are less than 1 mm in diameter and in which no cartilage exists, there are no gland or goblet cells.
- 4 The *smooth muscle*. Smooth muscle occurs in fairly large amounts in the mucous membrane under the epithelium. Generally the smooth muscle is arranged in two or three ribbonlike layers, the thickest being the muscle layer of the mucosa. The muscle fibers are located transversely in the membranous wall of the trachea and in the primary cartilaginous bronchi. The contraction of these muscle fibers narrows the lumen. Between the C-shaped cartilages there are longitudinal fibers crossed with the transverse oblique and longitudinal fibers.

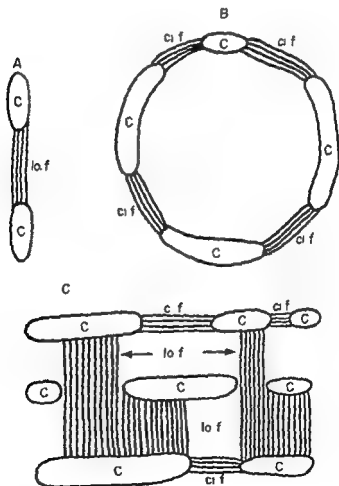
In the secondary cartilaginous bronchi the muscle is rather circular. The circular muscle is especially condensed around the orifices of the lobar bronchi and superior segmental branches of the lower lobes. Many circular muscle fibers are found in the walls of the terminal bronchioli. Their contraction may considerably narrow the bronchiolar lumens and cause severe dyspnea, e.g. in bronchial asthma. Further, they may occlude the bronchioli and so called "reflex atelectasis" may occur.

- 5 The *elastic elements*. These elements resemble fibers lamellae or granules. When the stretched elastic fibers are relaxed they return to their original length. According to location six different kinds of elastic fibers or layers are known. The most important are the inner elastic layer of the mucosa and the layers connecting the cartilages. However elastic fibers lamellae and granules exist almost everywhere in the bronchial wall, i.e. between the muscle fibers and the cartilage cells and also in the connective tissue around the glands.

The inner elastic layer is composed of longitudinal and circular fibers connecting the cartilages. Their location is shown in Figure 3. The longitudinal fibers limit elongation and the circular fibers limit the

dilatation of the bronchial wall. The elastic fibers also play a role in the pathology of bronchiectasis.

6. *The diverticula* The diverticula occur in the secondary and tertiary bronchi about four in a 1 cm length of bronchus. A diverticulum like the ducts of the bronchial glands opens on the surface of the mucosa and generally bores through the muscular layers sometimes penetrating as far as the peribronchial connective tissues. In the tissue surrounding a diverticulum an accumulation of lymphocytes can be observed. The physiologic function of these structures is unknown.



however pathologically they may play a role in the development of deforming bronchitis

### Summary

The tracheobronchial system may be called the "lung tree" but is referred to in this book as the *bronchial tree*. It consists of the trachea (the "trunk" of the tree) bronchi ("branches") and primary lobuli (the "leaves"). The exchange of oxygen and carbon dioxide takes place in the primary lobuli which are made up of the alveolar ducts and alveoli.

The terminology used to describe or to refer to the bronchial tree and its parts is in accordance with the international nomenclature agreed upon in London in 1949. The right and left lungs are each divided into lobes which in turn are subdivided into segments and in some segments into subsegmental branches.

The bronchial wall consists of four main structural parts: the trachea, the primary and secondary cartilaginous bronchi, and the intralobular bronchi. The bronchial wall has four layers: the mucosa, submucosa, fibrocartilaginous layer, and peribronchium. Six other important components are the layers of the mucosa—the epithelium, the pulmonary tonsil, the bronchial glands, the muscles, the elastic fibers, and the diverticula.



# Physiology

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## PHYSIOLOGY OF THE BRONCHI

The tracheobronchial system is the passage for airflow. During tests of normal respiration only about two thirds of the inhaled volume of a single breath enters the alveoli; one third ventilates only the conductive airways. In the respiratory tract the inhaled air is warmed and the humidity increased by the moisture of the bronchial secretion; thus the inhaled air is prepared for the alveoli.

The bronchial system has a cleansing function as well. The most important mechanism for this purpose is the action of the ciliary epithelium with the cilia moving incessantly day and night. With slow movements like a swan's head they withdraw then quickly strike forward (Fig. 4A and B). These movements sweep the dust granules from the surface of the mucosa toward the pharynx. However the granules do not touch the cilia directly because they are covered with a very thin invisible mucous carpet (Fig. 4C) on which the granules fall and float on the surface of the ciliary wave toward the pharynx to be swallowed into the stomach. The mucous carpet pulls the dust granules from areas on which no ciliated epithelium exists such as the vocal cords and the pulmonary tonsil. A severe inflammation destroys the cilia but they regenerate within 10 to 15 days. Cold and dryness hinder the movement of the cilia but their movement is accelerated in alkaline and salty media; therefore inhalation solutions contain small amounts of salt and sodium bicarbonate.

The bronchi undergo physiologic movements during respiration. These are torsion, angulation, elongation and caliber change.

**Torsion.** The bronchus rotates literally with each inspiration and returns with expiration like a torsion bar.

**Angulation.** In inspiration the bronchi parallel to the diaphragm increase the angle of their relationship to each other more than the diagonal bronchi. The magnitude of the increased angulation is from 2 to 15 degrees.



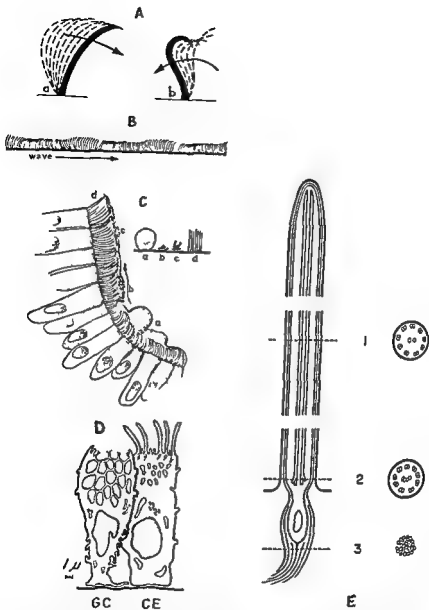


FIG. 4 A The movement of a cilium *a* the effective stroke and *b* the withdrawing of the cilium B The ciliary wave C On the right the sizes of *a* an erythrocyte *b* cocci and *c* bacilli in relation to the size of *d* the cilium On the left erythrocyte cocci and bacilli floating in the mucous carpet of the cilia (From Proetz<sup>20</sup>) D 1 (CE) in the tracheal structure and cross microscopy of the tracheal

**Elongation.** During normal inspiration, the larynx sinks 1 mm and the tracheal carina from 10 to 21 mm, because of the descent of the diaphragm. Also, as a result of the diaphragm's descent and the expansion of the chest cavity, the bronchi become elongated. These movements are greatest near the diaphragm and the posterolateral chest walls, they are smallest near the mediastinum and costovertebral sinus. This could be the anatomic and physiologic explanation of paravertebral pneumonitis.

**Caliber Changes.** The bronchial caliber increases in inspiration and diminishes in expiration. Normally, these movements are moderate in adults, but in children are always more pronounced because of the softer bronchial tissues. The bronchi move independently of respiration, because they move with the heart's pulsation. Naturally, the bronchi nearer the heart pulsate more than those further away.

The bronchologist observing and knowing the normal physiologic movements of the bronchi, recognizes pathologic processes in the chest cavity by increased, decreased, or restricted bronchial movements. When large mediastinal tumors, cysts, etc., exist, the movements of the chest wall are transmitted extensively to the bronchi. The movements change the caliber, especially, and are more extensive in pulmonary emphysema and bronchial asthma, because active expiratory pressure is exerted in these diseases. (Normally, expiration is passive, because after active inspiration, the thoracic organs return only passively to their normal resting position.) Thus active expiratory pressure affects the tracheal carina as well. The tracheal carina collapses in expiration and elongates in inspiration in an accordion-like action. The bronchial movements are diminished or absent if a malignant tumor embraces or covers the bronchial wall. In the case of an aortic aneurysm the lower portion of the trachea and the left main bronchus pulsate strongly.

## PHYSIOLOGY OF RESPIRATION

As previously discussed, the tubular system of the conducting airways—the nasal and pharyngeal cavities and the interior of the larynx, trachea, bronchi, and bronchioles—only conducts, humidifies, warms, and cleans the air. Since no effective gas exchange occurs in this system, it is called the *anatomic dead space*. In healthy individuals the volume of this space is almost identical with that of the *physiologic dead space*, which includes, besides the anatomic one, two additional types of volume: (1) the volume of gas which ventilates the alveoli with no capillary blood flow, and (2) the volume of gas that ventilates some alveoli in excess of that required to supply the capillary blood flowing around them with sufficient oxygen. Naturally these additional volumes occur to a significant degree only in pathologic pulmonary conditions.

Inhaled air is a mixture of inspired air and dead space gas rich in carbon dioxide and low in oxygen from the previous expiration. Further, air entering only the anatomic dead space on inspiration (with normal breathing, about one third of the inhaled air) does not come in contact with alveoli and does not contribute to the arterialization of the venous blood. Thus, the anatomic dead space, while it prepares the air for the alveoli by warming, humidifying, and cleaning, hinders the effectiveness of respiration by mixing and retarding the newly inspired air. These two disadvantageous factors are unimportant in healthy individuals, but in a patient with severe cardiopulmonary disorders every molecule counts. In these cases, the dead space of the nasal, pharyngeal, and laryngeal cavities can be eliminated by tracheotomy.

The air passes into the alveoli through the conducting airways only if the air pressure in the alveoli is less than the external atmospheric pressure (the air flows from a region of higher pressure to one of lower pressure). Conversely, after this inspiratory decrease in pressure (the expression "negative pressure" is frequently misused), the alveolar pressure has to be increased above the atmospheric pressure in order to press the air out of the lung.

### *Inspiration*

During inspiration a muscular effort enlarges the thoracic cage and so, decreasing the pressure, aspirates air into the alveoli. The muscles which dilate the thorax are the diaphragmatic and intercostal muscles, the levator costarum muscle, the serratus posterior superior and posterior inferior muscles, plus the so called "accessory respiratory muscles" (pectoral and sternocleidomastoid).

On inspiration, the highly arcuated dome of the *diaphragm*, by contraction of the radiant muscle fibers, becomes flattened. This action is similar to what happens in the barrel of a syringe when the needle, pointing upward, is drawn down. Contraction and flattening of the diaphragm open the phrenicocostal sinus. Its costal and diaphragmatic walls are in contact on expiration and in resting position, but they separate on inspiration and allow the inferior margin of the lungs to slide into the opened sinus (Fig. 5). Because of the descent of the diaphragm, the abdominal anterior wall bulges slightly forward. This part of breathing is called *diaphragmatic*, or *abdominal*, *respiration*. The abdominal breathing is more pronounced in men than in women. In healthy conditions this aspect of the breathing action is hindered only during pregnancy.

In inspiration, contraction of the previously mentioned muscles lifts the sternocostal hoop around the costovertebral joint and widens the chest cavity in both sagittal and frontal diameters. During this movement, naturally, the anterior extremities of the ribs perform the largest excursion.

The thorax widens in the sagittal diameter during inspiration because the anterior extremities of the ribs lie on a lower level than the posterior therefore when the ribs are elevated their anterior ends are thrust forward. The thorax widens in the frontal diameter for two reasons (1) The middle (lateral) portion of a rib lies at a level below the plane which passes through the anterior and posterior extremities and (2) each rib forms a curve which is larger than that of the rib immediately above therefore the transverse diameter of the thorax increases when the ribs are raised to a higher plane. This aspect of the breathing process is called *thoracic respiration*.

Insertion of the diaphragm is found anteriorly (*pars sternalis*) on the xiphoid process laterally (*pars costalis*) on the seventh to eleventh ribs and posteriorly (*pars lumbalis*) on the twelfth vertebra. On the sternal and costal parts during inspiration double excursion is performed by these insertions. In the course of elevation of the thoracic wall a diaphragmatic insertion on a rib moves upward and in the course of dilatation it moves outward (Fig 5). The diaphragm descends on inspiration.

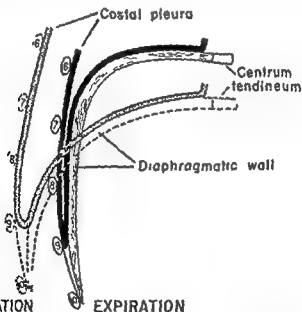


FIG 5 Costal and diaphragmatic wall of the pleurothorax are in contact on expiration but they separate on inspiration and all over the inferior margin of the lungs to allow the pleural cavity to expand. During the period of respiration the diaphragm contracts and moves downward and also expands. The diaphragm contracts due to the fact that its anterior and lateral insertions are elevated. The anterior call this phenomenon the pleural pump action.

despite the fact that its anterior and lateral insertions are *elevated*. The author suggests that this paradoxical phenomenon be called 'physiologic paradox motion'. This phenomenon is important in cases of pathologic paradox movements of the diaphragm—in cases of pneumothorax, valvular emphysema, and phrenic paralysis (see Chap. 3).

From the inspiratory elevation of the anterior and lateral portions of the diaphragmatic insertions, which counteracts the descent of the diaphragm, and also from other factors, it can be concluded that dilatation of the anterior part of the thorax is relatively less than that of the posterior. Actually, the dorsolateral part of the diaphragm performs the deepest descent, which is in accordance with the shape of the lungs.<sup>22</sup> Anteriorly less pulmonary tissue exists than posteriorly, because most of this area of the chest cavity is occupied by the heart and also by the diaphragm in its higher position. On the left side a negligibly small portion of the lingula is in contact with the diaphragm, but on the right side the middle lobe sits on a broader base. Very characteristically Christiaen<sup>10\*</sup> called this

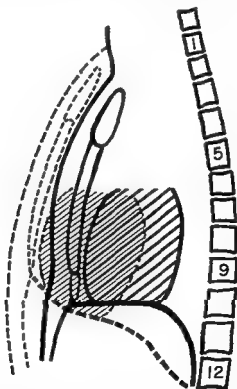


FIG. 11 During inspiration the heart is markedly moved forward, but descends on slightly because of the physiologic paradox motion (see Fig. 5)

base the "sole of the middle lobe." This disadvantageous location of the middle lobe and supposedly its restricted breathing capacity may be among the causative factors of the frequent occurrence of a middle lobe atelectasis, or middle lobe syndrome (see Chap 3).

The two motions inspiratory elevation of the thoracic wall and descent of the diaphragm, almost equalize each other in the anterior lower middle portion of the thorax therefore while the heart is markedly moved forward it descends only slightly on inspiration. The heart rests on the least moved spot of the diaphragm where its function is almost undisturbed (Fig 6).

### Expiration

The increased expiratory pressure is the result of the following factors are (1) the elastic resistance of the pulmonary tissues (2) the resistance of nonelastic tissues (costal cartilages and ligaments) and (3) the resistance of the respiratory muscles. After active inspiratory muscular action has ceased the organs are forced to return to their resting positions. Potential energy created during inspiration is stored in the stretched elastic tissues and the relaxation of the respiratory muscles causes the lung and thorax to return to the resting respiratory level. Thus under normal conditions the expiration is a passive process and it will be completed in 3 seconds. The time required for the active inspiration normally is about 1.2 times longer than that for the passive expiration. Under certain conditions such as coughing, sneezing, blowing the nose or playing wind instruments or in some cases of enforced expiration (e.g. in expiratory dyspnea) the expiration is also an active process produced by contractions of the internal intercostal muscles, the transversus thoracis muscle and the abdominal muscles.

The range of movement of the diaphragm between deep inspiration and maximum expiration averages 30 mm on the right side and 25 mm on the left. In quiet respiration the average movement is 12.5 mm on the right side and 12 mm on the left.

It is a well known fact that during inspiration the mediastinum becomes tightened and elongated. This phenomenon is observable with fluoroscopy. It is caused by two factors (1) The descent of the diaphragm and the forward placement of the sternum elongate the mediastinum in both cranio-caudal and dorso-ventral directions, and (2) the inflation of the pulmonary tissues more radiopaque. During expiration the air content of the lungs diminishes. This makes the mediastinal shadows broader. In crying infants during the extremely prolonged expirations the lung fields become strikingly obscured. Sometimes it seems as if the mediastinal

num reaches the lateral chest walls. This extreme air diminution may be recognized with percussion and may lead to a misjudgment. Therefore with crying infants great vigilance must be observed in evaluating the evidence obtained by percussion.

### *Interpleural Fissures*

A brief mention should be made of the important role of the interpleural fissures. These are only imaginary, because the atmospheric pressure affecting the entire huge alveolar surface and external chest wall forces the adjacent pleural sheets together. Despite this immense double-sided force, the pressure in the imaginary pleural fissures is less than 1 atm because the stretched pulmonary parenchyma, rich in elastic elements, engages traction on the visceral pleura. In quiet conditions the difference between the atmospheric and interpleural pressures is minus 2 to 4 mm Hg. At the end of deep inspiration it may climb to minus 15 to 30 mm Hg. This suction rises during inspiration in the interpleural fissures and thorax to overcome the air friction—physiologic resistance in the conductive airways. This increase in inspiratory low pressure also produces suction of the major veins, facilitates the blood flow toward the heart, and thus facilitates the blood circulation in the whole body.

### *Pulmonary Volumes*

**Classification and Tests** In the past pulmonary function has been studied only in research laboratories and its evaluation has been restricted to appraisal of the patient's history, physical signs, and chest x-ray findings in clinical work. Recently, with improved instrumentation and clinical methods, research tests have become a part of the clinical routine. However, it must be emphasized that pulmonary function tests do not supplant other diagnostic procedures. They measure only function and cannot replace an expressive history, physical examination, and radiologic, bacteriologic, bronchoscopic, or pathologic studies. A pulmonary disease may exist without disturbing function, and function tests may show normal values in the presence of a pulmonary lesion.

The lungs contain certain volumes of air during the different phases of respiration. For decades the tests of pulmonary function consisted of the measurements of these pulmonary air volumes. These tests failed to evaluate the functioning of the lungs because they measured only static values and the functioning of the lungs is a complex dynamic process. However, since alterations in volume may be caused by changes in function, and vice versa, it is important to know how volumes are classified and what their normal values are. Figure 7A illustrates the four primary lung volumes which do not overlap, and Figure 7B the four pulmonary capacities which include two or more primary volumes.

The *total capacity* and its subdivisions (*inspiratory capacity*, *expiratory reserve volume*, and *tidal volume*) can be measured by a simple spirometer (Fig. 9). During the test, the patient inspires maximally, and then expires completely into the spirometer. No time limit is imposed in this test.

The *residual* and *functional reserve volumes* are usually measured together by indirect methods, such as the *open* and *closed circuit* methods. In both these methods, gases which are not dissolved readily in the blood or the lung tissues are used. In the *open circuit* method, the nitrogen concentration (80 per cent) is known in air and lung, the patient repeatedly inspires pure oxygen and expires into a spirometer, which has been thoroughly rinsed with oxygen and contains no nitrogen. In a healthy individual, it takes about 2 minutes for the nitrogen to be completely rinsed out of the lungs into the spirometer, but in patients with poor ventilation (asthma, emphysema, cysts), it requires 7 minutes or more.

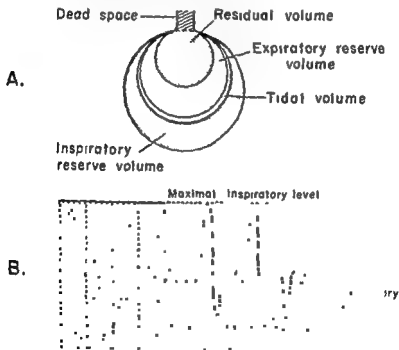


FIG. 9. A. Diagram of lung volumes. B. Spirometer tracing.

expiratory reserve volume (After Lammie et al.<sup>12</sup>)



In the *closed circuit* method the patient breathes into a spirometer in which the nitrogen concentration is zero and rebreathes until the mixing is complete. In both methods the residual volume or the total lung capacity can be easily calculated from the total amount of gas and nitrogen concentration in the spirometer. The residual volume test begins at complete expiration and measurement of the total lung capacity starts at the moment of maximal inspiration. The residual volume is obtained by subtracting the vital capacity which of course must also be measured from the total lung capacity. Determination of the same volumes may be carried out with helium. The functional reserve capacity may also be measured in the body plethysmograph.

The *total lung capacity* is usually obtained by measuring and adding together the functional residual volume and the inspiratory capacity.

TABLE 1 AVERAGE NORMAL LUNG VOLUMES  
(In liters)

	50 young men (recumbent)	50 young women (recumbent)
Age (years)	22.9	23.1
Height (cm)	176.2	163.4
Weight (kg)	73.5	57.2
Inspiratory capacity	3.79	2.47
Expiratory reserve volume	0.98	0.73
Vital capacity	4.78	3.14
Residual volume	1.19	1.10
Functional residual capacity	2.18	1.82
Total lung capacity	5.97	4.24
RV/TLC ratio	19.8%	25.9%

SOURCE: N. L. Haldreder, W. W. Fray and H. Z. Hyde. Effect of age on total pulmonary capacity and its subdivisions. *Am Rev Tuberc* 37:662, 1938.

The deviations from the average show that variations are considerable; consequently deviations from "normal" must be sufficiently large before they can be regarded as subnormal. Deviation in *functional capacity* may be as much as 20 per cent, for example.

**Pathologic Deviations.** *Low vital capacity* is only a sign and not pathognomonic of any disease; it can even appear without disease. It has been frequently noted in an *age in an* *erythremia* test; therefore a vital capacity test of no diagnostic value is not a good test. *Serial vital-capacity* test is in bronchial diseases, *vital capacity* may be disabled, and *an isolated* *pol*.

volvement pulmonary congestion and many other cardiopulmonary disorders

Increase of *functional residual capacity* represents hyperinflation which may result from emphysema peribronchiolar fibrosis conditions after pulmonary resections with compensatory emphysema deformity of the thorax etc The increase of *residual volume* means that the lungs remain inflated even after maximal expiration the patient cannot force his lungs back to normal size In these conditions pathology caused by bronchial obstruction due to secretion congestion edema or other lesions has developed in the thoracic wall in the respiratory muscles or in the lung tissues The residual volume may be decreased in cases in which alveolar occlusion appears in many portions of the lungs as in diffuse fibrosis

Changes in the residual volume/total lung capacity ratio increase when either the residual volume is increased (as in cases of asthma or emphysema) or the total lung capacity decreased (as in cases of fibrosis or pulmonary congestion) However residual volume greater than 35 per cent of the total lung volume does not mean pulmonary disability Older persons may be asymptomatic with a ratio as high as 50 per cent

The total lung volume is decreased in cases of severe pulmonary diseases (tumor atelectasis fibrosis edema exudate etc) in cases of collapsed pulmonary tissues (pneumothorax congestion emphysema etc) or when the expansion of the thorax is impeded (as in poliomyelitis extreme fibrosis etc) The total lung capacity may be normal or even increased in cases of emphysema large cysts or fibrosis if they lead to hyperinflation

### *Pulmonary Function*

**Tests** The pulmonary volumes are static factors but the pulmonary function with the gas exchange as its primary purpose is a dynamic complex process involving several other factors (see Fig 8) (1) alveolar ventilation which includes both volume and distribution of air adequate volume of the inhaled air must contact the alveoli each minute and the air must be distributed evenly to the several hundred million alveoli (2) capillary blood flow which also must be sufficient in volume per minute and distributed evenly to the alveoli and (3) diffusion by which oxygen and carbon dioxide pass through the alveolar capillary membranes

1 Alveolar ventilation is influenced by three components (a) the rate of breathing (b) the tidal volume and (c) the respiratory dead spaces

The rate or frequency of breathing is measured routinely in practice and hospitals It averages about eleven to sixteen times per minute in healthy individuals

The tidal volume means the amount of gas inspired or expired during

each respiratory cycle. Tidal volumes are seldom measured in clinical work, although they can be tested by spirometer or gasometer. In these tests it is more desirable to use air, because the oxygen may change both tidal volume and frequency. In both methods the volume of gas breathed per minute and the frequency must be measured, and from these measurements the average tidal volume can be calculated. In a healthy man, the tidal volume averages 500 to 600 ml with considerable individual variations.

In many laboratories it is difficult to measure anatomic dead space; therefore the use of average values is permitted. Approximate average values measured in recumbent position are about 160 ml in young men, in young women, 120 ml, and in older men, about 180 ml.<sup>15</sup>

From frequency, tidal volume, and anatomic dead space, minute volume and alveolar ventilation can be calculated. The average value of the alveolar ventilation for a resting healthy man is approximately 2 to 2.5 liters/min/sq m of body surface.

Effect of the dead space on alveolar ventilation per minute is demonstrated by the examples in Table 2. Dead space volume and minute volume were constant. In each case the same amount of air (8 000 ml) entered the airways, but not all of it entered the alveoli. In subject A, the high frequency (four times higher than in subject C) could not compensate for the low tidal volume (four times lower than in subject C), because in

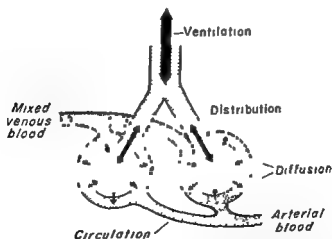


FIG. 8 The ventilation through the conducting airways (large arrow indicates (two smaller arrows) the gas diffusion and pulmonary blood flow entering the lung as arterial blood (light). Adequate distribution of both and unimpaired diffusion are necessary for normal pulmonary function. (After Comroe et al.<sup>12</sup>)

TABLE 2 DEAD-SPACE EFFECT ON ALVEOLAR VENTILATION PER MINUTE  
(Volumes in milliliters)

Subject	Type of respiration	Tidal volume	Rate	Minute volume (TV $\times$ rate)	Dead space volume	Alveolar ventilation on volume [(TV - DSV) $\times$ frequency]
A	Rapid shallow	250	32	8 000	150	3 200
B	Normal	500	16	8 000	150	5 600
C	Slow deep	1 000	8	8 000	150	6 800

subject A the same volume of the inspired air was mixed thirty two times and in subject C only eight times with the same volume of dead space gas. The alveolar ventilation per minute was as little as 3,200 ml in subject A and as much as 6 800 ml in subject C. Naturally the volume of inspired air which enters the alveoli is what counts and not that which enters the airways if the breathing is shallow a great portion of it ventilates only the dead space and not the alveoli.

Uneven ventilation can be tested by several methods (single- or multiple breath open and closed-circuit) using insoluble gases such as nitrogen or helium. Well and poorly ventilated areas are exposed by fairly constant or changing concentrations of these gases during one breath or several breaths. In healthy conditions the insoluble gas concentration does not rise more than 1.5 per cent in the first 500 ml of expired alveolar gas.

2. In many cases in evaluating pulmonary function distribution of the capillary blood flow is as important as those of alveolar ventilation but their measurement is more difficult. Recently rapid analyzers have been used for continuous recording of the carbon dioxide in expired alveolar gas from which the ventilation blood flow ratio (circulatory output) can be determined. In the healthy resting man the ventilation blood flow ratio is 4.5 or 0.8 (alveolar ventilation is about 4 liters per minute and blood flow is about 5 liters per minute).

3. Even if alveolar ventilation and the ventilation blood flow ratio test show normal conditions hypoxemia may occur because of impairment of diffusion.

Diffusing capacity can be measured by carbon monoxide or oxygen methods. The carbon monoxide method is simpler but it has the disadvantage among many others that it is not a physiologic gas. Since oxygen is a physiologic gas it is more suitable but the oxygen method is more complicated. The normal quantity for diffusing capacity in the resting individual is greater than 15 ml of oxygen mm Hg. During exercise this value increases in young men which is probably due to dilatation of the

patent vessels or to opening additional ones, but decreases in older individuals probably because of the reduction in the number of capillaries

All these dynamic processes of pulmonary function (ventilation blood flow, diffusion) aim to maintain the normal  $P_{O_2}$  and  $P_{CO_2}$  for arterialization of the venous blood. Elimination of  $CO_2$  is very important in the regulation of the acid base balance. Therefore, examinations of arterial oxygen, carbon dioxide, and pH yield valuable data, whether the function of the lungs is adequate or not. Estimation of arterial  $O_2$  from cyanosis of the patient is far from precise, because blueness of the skin is influenced by many factors (local cyanosis, anemia, different pigmentation of the skin, visual ability of the physician, etc.). An average physician is unable to detect reduced arterial  $O_2$  saturation by observing cyanosis until it falls to 75 to 85 per cent. With chemical or photometric methods, the  $O_2$  saturation may be measured or arterial  $O_2$  tension, which is more sensitive for recognizing a cardiopulmonary disease than the  $O_2$  saturation, can be tested by a bubble equilibrium technique. In healthy young men, the normal mean value of saturation of hemoglobin with  $O_2$  is 97.1 per cent, and the normal mean value of the arterial  $O_2$  tension is 95 mm Hg.

The ventilation is regulated chemically by the  $O_2$  and  $CO_2$  pressure and by the respiratory acidosis which increases the  $P_{CO_2}$ . All three may correct inadequate alveolar ventilation. Decrease in arterial  $P_{O_2}$  (hypoxemia) increases tidal volume and accelerates breathing rate. These changes are initiated in the chemoreceptors of the carotid and aortic bodies. However, they usually function only when hypoxemia becomes severe, that is, in an emergency. The respiratory center is extremely sensitive to changes in arterial  $P_{CO_2}$ . If it rises, the respiratory center responds with an increase in tidal volume and alveolar ventilation and later also in the rate of breathing. It is also well known that the sensitivity of the respiratory center is easily depressed by cerebral injuries, anesthesia drugs (morphine, barbiturates), severe hypoxemia or by high concentration of  $CO_2$  itself. A decrease in pH (acidosis) also can cause an increase in tidal volume and alveolar ventilation, probably due to involvement of both the centrum and chemoreceptors.

Respiration is also regulated by the vagal nerve endings in the pleura, bronchi and parenchyma. They are stimulated by stretching of the pulmonary tissue on inspiration; therefore, a reflex induced by this stimulus regulates the depth of the inspiration. This phenomenon is called *vagal stretch*, or *Hering Breuer reflex*.

Diagnostically, the measurements of arterial  $CO_2$  and pH may be as important as that of blood  $O_2$ . Arterial  $P_{CO_2}$  can be calculated from values of  $CO_2$  content, pH and  $O_2$  capacity, or by direct method of bubble equilibrium techniques. The normal value of arterial  $P_{CO_2}$  is about 41 mm Hg and that of plasma pH about 7.4.

TABLE 3 AVERAGE VALUES OF THE MOST IMPORTANT PULMONARY FACTORS IN TESTS IN YOUNG HEALTHY MEN

Breathing frequency	11-16/min
Tidal volume	500-600 ml
Dead space volume	150-160 ml
Alveolar ventilation	2-2.5 lters/min/sq m of body surface
Rise in gas concentration in even ventilation*	<1.5%
Ventilation/blood flow ratio	4.5 or 0.8
Diffusing capacity	15 ml O <sub>2</sub> /min/mm Hg
Arterial saturation of hemoglobin	97%
Arterial P <sub>O<sub>2</sub></sub>	95 mm Hg
Arterial P <sub>O<sub>2</sub></sub>	41 mm Hg
Plasma pH	7.4
Maximal breathing capacity	82-144 lters/min

\* In a test for even ventilation using the single-breath method the N<sub>2</sub> concentration does not rise more than 1.5% in the last 500 ml of expired alveolar gas.

**Pathologic Deviations.** Deviations from "normal" breathing rate may call attention to cardiopulmonary disorders but do not determine the magnitude of alveolar ventilation. The tidal volume alone does not determine the alveolar ventilation either but together with the rate of breathing may be helpful in determining massive hypoventilation of the lungs.

A physician can determine by inspection whether the thorax performs uniform and adequate excursions and measuring with tape he can even find more precise data of the chest excursion. By percussion he examines the excursion of the diaphragm and by auscultation whether the alveolar expansion is reasonably adequate. An anesthetist estimates the breathing capacities from the excursion of the anesthesia bag. A roentgenologist can determine that an eventual hypoventilation is due to a thoracic or diaphragmatic involvement. A physician who knows the importance of the alveolar ventilation and does not rely only upon the breathing rate tries with these examinations to find useful impressions of the pulmonary function. However, when any suspicion arises about the sufficiency of alveolar ventilation quantitative testing is necessary. Alveolar ventilation as was mentioned can be calculated from frequency, tidal volume and dead space volume.

Uneven ventilation is observed commonly by every physician when breathing sounds are diminished or absent regionally. By the roentgenologist when only some areas of the lungs "light up" during inspiration or by the surgeon in examining the chest opening. However, not every physician is familiar with the fact that uneven ventilation can be caused by changes in elasticity, bronchial stenosis, valvular emphysema, disturbance in expiration, etc. All these may involve a total lung or scattered

areas of one lung or both lungs. Hypoventilation of a whole lung causes hypoxemia, increased  $P_{CO_2}$ , and acidosis. Uneven ventilation leads to hypoxemia, but does not necessarily increase  $P_{CO_2}$ .

Impairment of diffusion may be secondary to a number of pulmonary diseases or due to the so called "alveolar capillary block," which has been found in cases of Boeck's sarcoid, beryllium granulosis, asbestosis, pulmonary scleroderma, alveolar-cell carcinoma, sulfur dioxide poisoning and diffuse metastatic lesions in the lungs.

Oxygen saturation may decrease in cases of hypoventilation, impairment of diffusion, venous to arterial shunts, or uneven ventilation.

Increased  $P_{CO_2}$  means hypoventilation, and its decrease means hyperventilation. However, normal or decreased  $CO_2$  pressure does not prove that a severe cardiopulmonary disorder is not present. In a case of severe disorder, the patient still may have capable alveoli and energy to hyperventilate, e.g., in cases of uneven ventilation, but it requires such hard respiratory work that pulmonary disability develops without inadequate  $CO_2$  elimination.

### *Tests of Mechanical Factors*

Ventilatory effort is reflected in changes of the interpleural pressure which are easily obtained by measuring intraesophageal pressures with a special balloon. The stiffness or distensibility of the lungs and thorax is called *mechanical compliance*. A normal value is 0.22 liter for each centimeter  $H_2O$  change in intrapleural pressure. Decreased compliance may occur in several pulmonary disorders such as congestion or fibrosis.

Another test is the measurement of the *work of breathing* which is related to the elastic recoil of the lungs, the resistance of airways, etc. The total lung resistance and the airway resistance are also measurable.

For evaluation of mechanical factors, the spiograph is generally used in clinical work. On a spiogram, tracings of simple breathing (tidal volume), forced inspiration and expiration (vital capacity and maximal inspiratory or expiratory capacity), and maximal breathing capacity (voluntary breathing capacity) are tested with a rapidly moving kymograph. A normal spiogram is shown in Figure 9. In measuring maximal breathing capacity (MBC) the patient breathes as deeply and as rapidly as he can for 15 seconds. The frequency is usually between 40 and 70 per minute, and the maximal inspiratory and expiratory capacity is about 50 per cent of the vital capacity tested without forced rapidity.

In cases of expiratory obstruction, e.g., in cases of emphysema, the lungs do not return after maximal inspiration (rapid overdistention) with a continuous movement to resting level. Between these two stages of respiration, several breaths are interposed, resulting in a steplike return

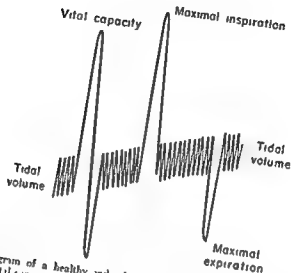


Fig. 9 Spirogram of a healthy individual. Tracing starts with normal breathing followed by vital capacity test, normal breathing, maximal inspiration, normal breathing, maximal expiration, and ends with normal breathing.

This phenomenon is called *air trapping*. It may also occur after maximal expiration.

### Bronchspirometry

Before lung operations it is definitely necessary to examine the functioning of each lung separately. Prior to irreversible collapse therapy and pulmonary resections it must be determined whether the alveolar surface of the remaining pulmonary parenchyma will be sufficient to maintain a normal oxygen level.

A separate functional examination of each lung is called *bronchspirometry*. This procedure which was introduced by Jacobson<sup>28</sup> in 1932 is carried out after adequate anesthesia by inserting a double lumen rubber tube (catheter) into the trachea. One tube ends in the trachea, the other in the left main bronchus. The lumens to the trachea and the left main bronchus are occluded with pneumatic rubber cuffs (Fig. 10). The left lung breathes through the longer tube and simultaneously and under shorter one ending in the trachea. In this way it is possible to determine the functioning of each lung separately and under identical conditions. Norris et al.<sup>29</sup> have used a single lumen catheter largely eliminating the undesirable effects of stenosed breathing often caused by the double tube. The lumen of the catheter provides an airway



to the left lung and air exchange to the right lung is maintained around the catheter. Of the double-lumen catheter, the Carlens' type is now the most widely used. There have been few attempts to attain new methods for examining the functions of lobes and segments separately.

With bronchspirometry it is possible to measure the tidal volume, vital capacity, and oxygen consumption of each lung separately with two recording systems. Normally the right lung performs about 55 per cent of the ventilation and of the oxygen consumption and the left lung 45 per cent of each. Noticeable deviation from this ratio means unilateral pulmonary disease.

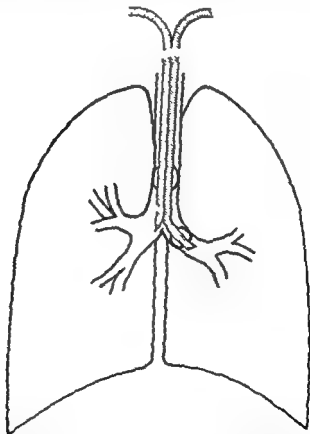


FIG. 10 Schematic drawing of the insertion of a bronchspirometric catheter. The right lung breathes through the stippled tube; the left through the striped.

## BRONCHOSTENOSIS

Diseases of the bronchi, lungs and other thoracic organs frequently cause bronchostenosis. No matter what the cause of stenosis, the result of the mechanical obstruction is the same. This chapter deals with these consequences.

The factors in the origin and progress of stenosis are known; they are anatomic, functional and etiologic.

*Anatomic*

The stenosis may be located in the trachea, in a main bronchus, in a lobar bronchus, in a segmental bronchus or in a minor bronchus up to the bronchioli. This is the anatomic factor. The general effect depends on the extent of the involved area supplied by the stenosed or obstructed bronchus. But the bronchi may narrow in groups, as may all the bronchioli (e.g. in bronchial asthma); the general effect then is the most severe.

*Functional*

Depending on the degree of the stenosis we observe simple stenosis, valvular emphysema or atelectasis. These are the functional factors.

Simple Stenosis (Bypass valve Mechanism)

men of the

the steno

the norm

narrowing

sound arises in the majority of cases. The sound in expiration the

wheeze "whistling"

Valvular

the bronchi

Atelectasis (Bypass Mechanism) In this the bronchus is closed in

both phases of respiration

*Etiologic*

The obstructive material or lesion may be located intrabronchially, endobronchially or extrabronchially. Thus bronchostenosis may be caused by any one of a series of different diseases. The underlying disease is the etiologic factor (Chap. 1).

Respiration is a dynamic process. During respiration the thoracic pressure varies. The serial pressure alterations influence the flow of the blood through the chest cavity and in this way affect the oxygen supply of the organism.

Bronchostenosis or occlusion disturbs this dynamic physiologic balance and creates a pathologic condition which may involve the whole organism.

In bronchostenosis the preparation of air for the alveoli may be inadequate if the accelerated breathing transports air so quickly to the alveoli that there is not sufficient time for warming and humidification. Cleansing of the bronchi and lungs and effective functioning of cilia are hindered. Secretion of the glands is restrained. Chevalier Jackson<sup>11</sup> calls this process the "self drowning of airways." Superimposed secondary infection may cause severe inflammation such as recurrent pneumonia or pleuropneumonia.

In bronchostenosis two possibilities may arise. (1) In spite of the degree of the stenosis the patient is able to keep the previous normal air exchange with increased minute volume. (2) Air exchange falls below the normal level and causes dyspnea.

Superficial observation does not yield adequate information about the patient's condition. For example, doubled respiratory rate cannot be recognized in a quietly sitting individual until it is tested, and an average physician is unable to detect reduced arterial oxygen saturation by observing cyanosis until it falls to 75 to 85 per cent. Exact observation and recording can be done only with a knowledge of the types and symptoms of dyspnea. It may be inspiratory, expiratory or mixed because of the impairment respectively of inspiration, expiration or both. Also two other types of dyspnea are known: reflex and physicochemical. Both may be observed at the same time.

**Reflex Dyspnea.** Reflex dyspnea is based on the Hering Breuer reflex. The most important stimulus of the respiratory center is increased carbon dioxide in the blood. But another stimulus exists as well: afferent impulses from sensitive nerve endings in respiratory muscles, chest wall, pleura and lungs. These impulses tend to limit both inspiration and expiration but especially the depth of inspiration in normal conditions. This is the Hering Breuer reflex.<sup>12</sup>

If after a suddenly occurring bronchial obstruction, sensation of normal airflow and normal pressure in the chest cavity rapidly change, afferent impulses signalize the pathologic condition to the respiratory center. Inceased respiratory reflex movements occur which may upset respiratory balance and urgently indicate surgical intervention.

A characteristic example of this process is presented by the panic of a young tracheotomized child which alone may defeat decannulation. When a child wears a tracheal cannula for a long period, he becomes accustomed to the fact that through the relatively wide tube, air enters the lungs without any effort. But after the cannula has been removed and the stoma collapsed or covered for a test of breathing, air passes through the physiologic but narrower canals of the nose and larynx. Thus altered

condition requires increased work from the muscles and alters intra thoracic pressure therefore afferent impulses signalize abnormality and reflex dyspnea occurs. In this condition the child breathes vigorously and partially collapses the weakened wall (at the stomal level) of the otherwise healthy trachea with normal caliber. The cannula must be replaced. Under such circumstances the child is not able to get rid of his cannula except by gradual adaptation sedation or under narcosis.

**Physicochemical Dyspnea** This is caused by a decrease of oxygen tension and an increase of carbon dioxide tension in the blood due to hypoventilation of the lungs. Such a progressive decrease in oxygen results in hypoxia. The patient tolerates this slowly increasing dyspnea if he does not exert himself. The body accommodates to the extreme decrease in the amount of oxygen metabolic processes diminish and muscular strength activity and vital level decrease. Men near freezing and hibernating animals exist in this condition which is called *ata parva* meaning *small life*. In the hypoxic state the respiratory center becomes accustomed to the stimulus of the greatly increased carbon dioxide pressure. The physician must be very careful with patients in this condition just as in the removal of persons from high atmospheric pressure. If the obstructed quickly disappears as a result of removal of obstructing material or by tracheotomy after some free breaths the high carbon dioxide level in the blood quickly decreases and the respiratory center does not receive its pathologically accustomed massive stimulus. This little shock may definitely paralyze the long depressed respiratory center which was already bordering on paralysis. In more favorable cases it may result in the suspension of respiration (apnea) from which the patient can be delivered only by simple or bronchoscopic carbon dioxide insufflation.

Hypoxia respiratory decompensation and its rapid progression are accurately determined by an oxygen saturation test of the blood. Unfortunately this test is not so simple that its use can be recommended for daily practice. Hypoxia is recompensed by minute volume increase and accelerated and superficial respiration therefore respiratory decompensation must be suspected if in laryngeal tracheal or bronchial stenosis the pulse and respiratory rate gradually or rapidly increase. Cyanosis is of no great importance. In chronic dyspnea the complexion of the patient is not livid or blue but remarkably pale perhaps when gray. Consequently the patient's complexion and general appearance is of great significance. A hypoxic patient appearing to be in a good condition while actually in more serious condition is able to take a short walk but the slightest overexertion may be fatal. A normal morphine dose excitement before surgery further narrowing of a tracheal stenosis by local anes thesia or clamping it may be enough to increase oxygen insufficiency and definitely paralyze the respiratory center.

The foregoing explanation relates to respiration the following will pertain to blood circulation. As previously mentioned intrabronchial and intratracheal pressure alterations affect the amount of blood flowing through the chest cavity. Every effective inspiration increases the pressure in the pulmonary arteries. Consequently in patients with laryngeal, tracheal or bronchial obstruction capillary blood pressure may increase and cause serum diffusion into the alveoli. However forced inspiration produces another effect. Increased suction aspirates more serum into the alveoli. As the effective alveolar surface diminishes inspirations deepen respiration accelerates and inspiratory suction and capillary pressure increase more and more. This vicious circle leads first to congestion later to drowning of alveoli and finally to pulmonary edema.

Thus pulmonary dyspnea results in a double effect respiratory and cardiovascular.

**Respiratory.** Moderate stenosis or obstruction in a minor bronchus causes compensatory dyspnea (reflex or physicochemical). Severe laryngeal or tracheal stenosis and obstruction of a main bronchus sooner or later result in hypoxia and respiratory decompensation.

**Cardiovascular.** Cardiovascular disturbances through congestion and drowning alveoli result in pulmonary edema and circulatory disturbances.

We have seen that bronchostenosis may cause profound alteration in bronchopulmonary physiologic action. This not only involves the distal parenchyma but also alters normal intrathoracic pressure, impairs its regulation, pathologically affects the amount of blood flowing through the chest cavity and alters the fluid content of the lungs. Thus irreversible changes may occur and it is therefore necessary to stop this dangerous process.

It must be determined with observation and accurate examinations whether or not a patient is able to maintain a sufficient oxygen level. Approaching or already present respiratory decompensation must be recognized and fatal consequences prevented by emergency oxygen in sufflation or surgical intervention.

Today a physician cannot be satisfied with evaluating a cardiopulmonary condition only by appraising a patient's history, physical signs or x-ray findings of the chest. In many cases quantitative measurements of the pulmonary volumes, alveolar ventilation, capillary blood flow and diffusing capacity, examinations of the arterial oxygen, carbon dioxide and pH and tests of the mechanical factors or some part of all these tests are necessary. Deviations from the normal may guide the physician to correct diagnosis and precise evaluation of his therapeutic result.

In pathologic respiratory mechanisms especially in cases of foreign bodies in children comparison of x-ray signs and the degree of dyspnea

yields very valuable data in localizing the stenosis. It is very important to keep in mind that severe dyspnea is caused (except for hypopharyngeal laryngeal or tracheal stenosis) only by simultaneous *bilateral* stenosis of the major bronchi. In a quiet state an infant or a child breathes well with one healthy lung (e.g. when the main bronchus of the other lung is occluded). Thus in stenosis of the lower respiratory tract if symptoms of severe dyspnea are observed laryngeal tracheal or bilateral stenosis or occlusion of major bronchi must be considered provided no other pathologic process exists. Further data may be obtained by x ray examination (Figs 15 and 113).

Airflow to and fro through a stenosis may produce *stridor*. Stridor may be heard as a blowing or whistling sound similar to a rattle, the roll of a drum, the sound of yawning, etc. Depending on the phase of respiration in which it is audible stridor may be inspiratory or expiratory as in dyspnea. It signals only stenosis and very rarely an occlusion.

In simple stenosis stridor can be heard chiefly during expiration because air strikes the obstacle and makes a sound (oral wheeze) (Fig 20). It is most audible when the examiner's ear is placed in front of the patient's mouth.

In complete valvular emphysema stridor can be heard only for some minutes after a quick occlusion, for example in foreign body aspiration. When the lung is already blown up to its maximal inspiratory volume sound ceases to exist because the small amount of air which enters to replace the absorbed air at the climax of inspiration causes no stridor.

In regular or irregular incomplete valvular emphysema stridor may be audible in inspiration (in expiration the bronchus is closed) or for a very short time after expiration when trapped air is escaping through the incomplete occlusion (Fig 17). This gentle stridor after expiration is audible only with the stethoscope or phonendoscope and sounds like an "after blowing" closely following the expiration. It is easily recognizable to the skilled ear because its tone differs distinctly from the sound of expiration. This symptom is important because roentgen symptoms may be absent if only a portion of the lung is involved. Some foreign bodies have been diagnosed only by means of this delicate symptom.

In a majority of the cases of atelectasis no stridor exists because the bronchus is closed in both phases of respiration.

In every case of bronchostenosis the author examines the patient with a stethoscope following the fork of the trachea and major bronchi on the anterior chest wall. By this examination if stridor is present the stenosis may be localized. The stenosis is located where the stridor sounds strongest. For examples see Chapter 3 Case 1 and Chapter 12 Case 9.

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yields very valuable data in localizing the stenosis. It is very important to keep in mind that severe dyspnea is caused, except for hyperextension of laryngeal, or tracheal stenosis only by some minor localized stenosis of the major bronchi. In a great state of emphysema or a child bronchitis with one healthy lung (even when the main bronchus of the other lung is occluded) Thus in stenosis of the lower respiratory tract dyspnea and even dyspnea are observed. Localized tracheal or bronchial stenosis or occlusion of major bronchi may be considered provided no other pathologic process exists. Further data may be obtained by a very careful examination (Figs. 15 and 113).

Airflow to and from through a stenosis may produce a whistle which may be heard as a blowing or whistling sound similar to a note of the flute or a drum, the sound of vawn etc. Depending on the phase of respiration in which it is audible stridor may be inspiratory or expiratory or both. It is usually only stenosis and very rarely an obstruction to the airway. In simple stenosis stridor can be heard chiefly during expiration but as air strikes the obstacle and makes a sound heard when the patient's mouth.

It is most audible when the examiner's ear is placed in front of the patient's mouth. In complete valvular emphysema stridor can be heard only for some minutes after a quick occlusion for example in a normal person a person. When the lung is already blown up to its maximal expansion volume sound ceases to exist, because the small amount of air which enters to replace the absorbed air at the climax of inspiration cannot escape.

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### Summary

The bronchial tree is the passage for airflow in which the inhaled air is warmed humidified and cleaned on its way into the alveoli

The bronchi undergo physiologic movements during respiration These are torsion angulation elongation and caliber change

No effective gas exchange occurs in the conductive airways which are called *anatomic dead space* The *physiologic dead space* includes besides the anatomic one two others which play a significant role only in pathologic pulmonary conditions

During *inspiration* a muscular effort enlarges the thorax in all three dimensions and aspirates air into the alveoli as follows

- 1 The dome of the diaphragm flattens and descends widens the thorax vertically opens the pleurocostal sinus and lets the inferior margins of the lungs slide into the opened sinus This process is called *diaphragmatic* or *abdominal respiration*

- 2 The sternocostal hoop is elevated which widens the thorax in both sagittal and frontal diameters This process is called *thoracic respiration*

- 3 The leaves of the diaphragm descend while its sternal and costal margins are elevated These processes counteract each other therefore this movement may be called the "physiologic paradox motion"

- 4 Dilatation of the anterior portion of the lungs is relatively less than that of the posterior The right middle lobe is located at a disadvantageous place in regard to its breathing capacity

- 5 The heart rests on the least moved spot of the diaphragm where its function is almost undisturbed by the respiratory movements

During *expiration* the stretched elastic tissues with their potential energy cause the lung and thorax to return to the resting respiratory level In normal conditions the expiration is a completely passive process and it becomes active only in certain conditions

In *inspiration* the mediastinum becomes tightened and narrowed in its transverse diameter because of its elongation in both vertical and sagittal diameters On *expiration* the mediastinum widens This widening is extremely pronounced in crying infants

In quiet circumstances pressure in the interpleural fissures is less than 1 atm and decreases further on inspiration This low pressure sucking blood from the caval veins into the heart facilitates blood circulation

The *lung volumes* are merely anatomic factors and tests of these do not evaluate pulmonary function However alterations in volume may be caused by changes in function and vice versa therefore alterations in volume can be of diagnostic value and signify changes in the course of a disease during therapy These tests are relatively simple

The primary function of the lungs is a single one *gas exchange* How

## PHYSIOLOGY

ever the pulmonary function is complex involving many other factors. Normal ventilation includes both adequate volume and even distribution of the air into hundreds of millions of alveoli. capillary blood flow also must be adequate in volume and evenly distributed. diffusing capacity of the lungs must be unimpaired for the pulmonary function to be considered normal.

All these dynamic processes aim to maintain normal  $P_{aO_2}$  and  $P_{aCO_2}$  for arterialization of the venous blood. The examination of the arterial oxygen, carbon dioxide, and pH may yield valuable data whether the respiration is adequate or not.

The mechanical factors are also of great importance. In some pathological cases adequate gas exchange may be achieved by a considerably increased work of breathing. Therefore increased breathing may be the only finding which indicates a cardiopulmonary disorder.

Quantitative measurement of all these factors requires a number of tests which can be simple or extremely complex. Deviations from the normal may guide the physician to a more correct diagnosis and to a better evaluation of the therapeutic result.

Before lung operations (irreversible collapse therapy, pulmonary resections) it is definitely necessary to examine the function of each lung separately. Separate functional examination of the lungs is called bronchopulmonary function tests. Marked deviation from normal ratio means unilateral pulmonary disease. Prior to pneumonectomy bronchopulmonary function tests are also in examining the function of the lung to be spared but this test is not precise enough to predict the postoperative capability of this lung. In this case measurements of pulmonary arterial pressure with unilateral arterial occlusion by means of cardiac catheterization may lead to better evaluation.

Diseases of the bronchi, lungs, and other thoracic organs frequently cause bronchostenosis. No matter what the cause of the stenosis the result of the mechanical obstruction is the same. Factors involved are anatomic, functional, and etiologic.

Bronchostenosis or occlusion may disturb the dynamic physiologic balance of the gas exchange not only by preventing adequate air from entering the alveoli but by accelerating the venous and retarding the arterial blood flow. Two types of dyspnea may arise: reflex based on the Hering-Breuer reflex and physicochemical caused by decrease of oxygen and increase of carbon dioxide tension due to hypoventilation of the lungs.

Pulmonary dyspnea results in respiratory and cardiovascular disturbances. Severe laryngeal or tracheal stenosis and obstruction of a main bronchus sooner or later results in hypoxia and respiratory decompensation. Cardiovascular disturbances through congestion and the drowning of the alveoli result in pulmonary edema and circulatory disturbances.

In evaluating the degree of these disturbances quantitative measurements of the pulmonary function are necessary in many cases. Comparison of the x ray signs and the degree of dyspnea may yield very valuable data in localization of a stenosis. Stridor may be of great value not only in localization but even in discovery of a stenosis.

# Roentgenology

## PATHOLOGIC RESPIRATORY MECHANISMS

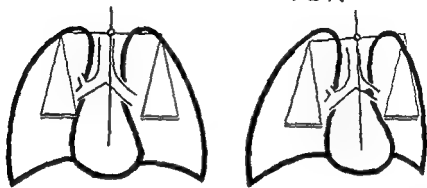
Normally changes of volume (expansion and reduction) of both lungs are well balanced therefore the mediastinum and the heart keep an unaltered position during respiration. The mediastinum and the heart are like the tongue of a pair of scales with the lungs as the plates except that the balance is of volume not weight. Immobility of the "tongue" indicates an equilibrium of the volume changes in both lungs (Fig. 11).

If the lumen of a major bronchus is substantially constricted or obstructed then the equilibrium of volume is upset and the "tongue" of the "scales" deviates. Thus pathologic respiratory mechanisms arise. A pathologic respiratory mechanism is caused by an obstacle to the expansion and reduction of volume of the lungs or a part thereof. This obstacle upsets the normal equilibrium of volume. It is marked by a lateral shift of the mediastinum and the heart (Fig. 11) or the x-ray *middle shadow*. This sign might be named the "scales sign".

Holznercht<sup>2</sup> a roentgenologist of Vienna in 1899 described an x-ray sign of bronchostenosis: *the inspiratory retraction of the mediastinum and heart into the affected chest cavity*. He observed this sign in a case of simple stenosis of the right main bronchus. In the past decades a number of writers have been dealing with the question of the pathologic movement of the mediastinum and heart. Some have stated that in cases of "bypass valve" action for which the term *simple stenosis* will be used in this monograph and in cases of atelectasis no pathologic movement of the mediastinum and heart is observable. The author's own observations have contradicted this. It was observed that Holznercht's sign appears in all three forms of pathologic respiratory mechanisms that will be discussed below (simple stenosis, valvular emphysema, and atelectasis). The mediastinum and heart *independent of the degree of the stenosis shift toward the involved side*.

As mentioned above and as described in Chapter 2, the pathologic respiratory mechanism appears in three forms: simple stenosis (bypass

## INSPIRATION



NORMAL

SIMPLE STENOSIS

FIG 11 The chest scales in which the 'tongue' of the scales is represented by the mediastinum and heart. Immobility of these structures indicates equilibrium of the volume of both lungs during respiration while their dislocation indicates a disturbance of equilibrium, that is to say, stenosis of a major bronchus.

valve mechanism), valvular emphysema (check-valve mechanism—ventil-stenosis, or obstructive emphysema), and atelectasis (stop valve mechanism).

All three forms can be caused by various sizes of the same broncho stenosis, but are substantially different in respect to the condition of the affected part of the lung. All three forms can be observed during the course of the same illness successively, alternately, or simultaneously, following the changes of the degree of the bronchostenosis (Case 2, in this chapter, Figs 14, 66, and 110). The physical state of the part of the lung distal to the stenosis is as follows:

In a simple constriction the lung or part of a lung which belongs to the affected bronchus gets less air during inspiration than the other lung or the rest of the affected lung. In valvular emphysema the involved part of the lung is inflated. In an atelectasis the blocked part of the lung is airless.

### Simple Stenosis

In simple stenosis the lumen of the bronchus is large enough to allow air to pass to and fro, but with a deep inspiration the lung distal to the constriction receives less air than the normal lung in the same space of time. The thorax dilates on both sides in the same proportion. Thus, the lung which gets less air and dilates less during inspiration is followed by the mediastinum and the heart to its own side, i.e., to the involved side. This occurs during inspiration, because the inspiratory action is generally faster

and more vigorous. The crying child for instance inspires in 1 or 2 seconds the air needed for the long expiration connected with weeping.

Even the slightest motion is of diagnostic value. The inspiratory tightening of the mediastinum is not to be considered as a Holzknecht sign.

The pathologic movement of the mediastinum is caused by an increased

tion is greater if the main bronchus or the bronchus intermedius is constricted. The fine lateral shift of the mediastinum can also be observed in children in stenosis of the major lobar bronchus. But a still smaller bronchial stenosis generally does not effect any motion of the mediastinum. The degree of a bronchial stenosis which causes a pronounced shift is demonstrated by the characteristic case of a two year-old child who had inspired the winding screw of an alarm clock. The foreign body was impacted into the orifice of the left main bronchus and constricted the bronchus in such a way that the left lung breathed through the hole of the foreign body (Fig. 12). The external diameter of the foreign body was



Fig. 12. A lateral x-ray film shows a winding screw of an alarm clock in the orifice of the left main bronchus of a two-year-old boy. The left lung was breathing through the hole of the foreign body. The external diameter of the screw, which indicated also the diameter of the lumen of the bronchus, was 7 mm and the diameter of the hole was 1.8 mm. A distinct Holzknecht sign was observed to the left because the lumen of the left main bronchus was reduced by about 75 per cent. In the left lower corner the removed foreign body.

7 mm and the diameter of the inner hole was 1.8 mm. The distinct Holzknacht's sign could be observed to the left.

Holzknacht's sign can be observed in its most pronounced form in children since it occurs more often, appears sooner and takes a more pronounced form. The tissues of the child are more elastic and generally more easily movable than the tissues of adults. There is less fixation in the thoracic cavity. The tissues are more expansile, thus making a more extensive deviation of the organs possible. The lumens of the bronchi are narrower than in adults; therefore even a small obstructive peg can produce pronounced symptoms.

In describing Holzknacht's sign for the patient's history, direction of the movement of the middle shadow is indicated during inspiration. Thus *Holzknacht's sign to the right* means that during inspiration the middle shadow shifts toward the right side. The description should be "toward" rather than "to" because for example in the case of a complete obstructive emphysema the mediastinum and the heart cannot get across to the uninvolved side.

To demonstrate the importance of the Holzknacht sign in simple stenosis the following case is most characteristic.

**CASE 1.** A girl two years of age was under clinical treatment for 45 days with the diagnosis of laryngeal croup. As the respiratory disturbances did not cease she was transferred in a severely dyspneic state to another hospital. There she received strophanthin, camphor, dextrose and penicillin for 12 days. Secretion was aspirated twice from the trachea. In the morning before she was transferred to our hospital bronchoscopy was performed. The child well developed was in a severe dyspneic state. From the drawing in of the jugulum and in the epigastrium and from the function of the accessory muscles one could unquestionably ascertain that the child was suffering from violent stenosis of the air passages. Fluoroscopic examination showed pronounced Holzknacht's sign to the left. Stridor found in examination by stethoscope was the strongest just below the larynx.

By direct laryngoscopic examinations a circular stricture in the trachea 1.5 cm below the glottis was perceived. The tissues at the point of the stenosis were reddish and moderately hard.

At this state of the inspection it could have been concluded that the stricture of the trachea which caused the severe state of the child was the consequence of diphtheria. But the fluoroscopic examination showed *Holzknacht's sign to the left*. This indicated a stenosis of the left main bronchus.

The bronchoscope was passed easily through the trachea and the 8 mm long and 3 mm wide skin of a sunflower seed was removed. Because of the stricture of the trachea tracheotomy was performed.

Two days later during the changing of the cannula the child coughed out through the cannula a lentil sized necrosed piece of tissue. Direct laryngoscopic examination showed that the stricture of the trachea had completely

disappeared 11 days after the operation. The cannula was removed. On the fifteenth day the child left the hospital in perfect health.

The child had not suffered from diphtheria. The piece of sunflower skin had lain with the convex surface against the wall of the trachea. It had been in the trachea 57 days and produced granulation stenosis. On the day of the operation the first bronchoscopic manipulation had pushed it into the left main bronchus where it had been found amid an entirely intact surrounding. The Holzkecht sign had helped to locate the foreign body and to clear up the condition. After removal of the foreign body the granulation tissue was coughed out and the child recovered.

### Valvular Emphysema (Ventilstenosis or Obstructive Emphysema)

The term *ventilstenosis* refers to a condition of the bronchus. The term *obstructive emphysema* refers chiefly to the condition of the lung. The term *valvular emphysema* comprises both meanings and is therefore more applicable and more expressive than the other terms.

It should be mentioned too that even the term *emphysema* is inaccurate because in cases of valvular emphysema destruction of the pulmonary elastic elements which is very characteristic of emphysema is absent. Furthermore the alveoli are in the stage of maximal inspiration and are inflated only during expiration. Therefore since this condition is pathologic during expiration it should be called *expiratory hyperinflation* only. However the term *emphysema* will be used here because it has taken root and been used for several decades.

Classification of valvular emphysema into four types is proposed by the author: complete, partial, irregular, transitory, and incomplete.

**Complete Valvular Emphysema.** In a complete valvular emphysema the bronchus is obstructed during expiration as well as during the pauses.



FIG. 13. Anteroposterior x rays of a nine-year-old girl with complete valvular emphysema due to a tuberculous lymph node which ruptured into the left main bronchus. A. During expiration. B. During inspiration.



of respiration. In fact it opens and allows air to penetrate into the closed part of the lung only when inspiration reaches the maximum. The closed part of the lung is inflated and dilated in consequence of the valvular action. In infants and children the expansion is greater because the tissues are less resistant. In older children and in adults the expansion is equivalent to the state of maximal inspiration.

During expiration and the subsequent pause in breathing the expanded lung pushes the mediastinum and the heart to the normal side and presses the diaphragm downward. It is then that the state of expiration should be photographed (Fig. 13A). The mediastinum and the heart are moving back to the normal position during inspiration in the direction of the involved side (Holtzknicht's sign) while the downward pressed diaphragm is ascending (paradox motion)\*. With the maximum of inspiration the middle shadow reaches the median line, thus it is in its normal position. This state is constant as long as the patient does not expel the inspired air. The state of inspiration then is photographed (Fig. 13B). Respiratory sounds cannot be heard on the expanded side.

Special roentgenograms during inspiration and expiration are desirable for the demonstration of every single case, because the normal lung is deficient in air during expiration and this could be considered as an atelectasis (Fig. 17). The state of deep inspiration shows the normal condition. The two roentgenograms together are of greatest importance in establishing the diagnosis and in demonstrating the condition (Fig. 112).

**Valvular Emphysema with Partial Mediastinal Shift.** Valvular stenosis occurs not only in a main bronchus but also in one or two lobar bronchi. In such a case chiefly the normal lobe of the involved side is compressed during expiration; this is the result of the inflation of the affected lobe. The affected lobe is emphysematous and the normal one is deficient in air (Fig. 14). Naturally, in these cases the moving of the mediastinum is less than when the whole lung is involved. The case on page 47 is of this type.

\* Paradoxical movement of the diaphragm appears also in two other conditions: pneumothorax and phrenic paralysis. In pneumothorax the mechanism is exactly like that of valvular emphysema, even though in pneumothorax the air is trapped in the chest cavity and in valvular emphysema it is trapped in the "lung tree." In cases of a unilateral phrenic paralysis the paralyzed leaf of the diaphragm ascends to a higher level into the chest cavity; in inspiration it ascends even higher because the sucking effect chiefly of the healthy side pulls it up. However there is this characteristic difference between valvular emphysema and phrenic paralysis: The suction of the healthy side which pulls the diaphragm up also affects the mediastinum and heart and in inspiration pulls them to the uninvolved side; the paralyzed diaphragm

**CASE 2** A boy three years of age had a poor appetite for 7 months and was losing weight. Two months before hospital investigation a right lateral segment shadow could be observed (epituberculosis) (Fig. 14A), probably a rupture of a lymph node into the lateral segmental bronchus.

A few days before investigation this shadow had been moving upward (Fig. 14B and C). An obstructive emphysema developed in the right middle and lower lobes with Holtzknicht's sign to the right. The motion extended chiefly to the heart (Fig. 14B and D). A new rupture into the stem bronchus was evident.

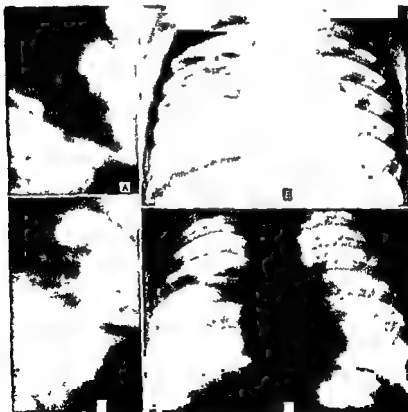


FIG. 14. Lateral x rays of a three year-old boy. A. Segmental atelectasis (epituberculosis) in the lateral segment of the right middle lobe due to rupture of a tuberculous lymph node into the corresponding bronchus. B. A new rupture occurring two months later into the stem bronchus causing valvular emphysema in the right middle and lower lobes. C. The emphysematous middle and lower lobes pushed the atelectatic segment upwards. X ray (B) taken on expiration x ray (D) taken on inspiration showing Holtzknicht's sign to the right (see Case 2).

of respiration. In fact, it opens and allows air to penetrate into the closed part of the lung only when inspiration reaches the maximum. The closed part of the lung is inflated and dilated in consequence of the valvular action. In infants and children the expansion is greater, because the tissues are less resistant. In older children and in adults the expansion is equivalent to the state of maximal inspiration.

During expiration and the subsequent pause in breathing the expanded lung pushes the mediastinum and the heart to the normal side and presses the diaphragm downward. It is then that the state of expiration should be photographed (Fig. 13A). The mediastinum and the heart are moving back to the normal position during inspiration in the direction of the involved side (Holtzknacht's sign) while the downward pressed diaphragm is ascending (paradox motion).<sup>\*</sup> With the maximum of inspiration the middle shadow reaches the median line, thus it is in its normal position. This state is constant as long as the patient does not expel the inspired air. The state of inspiration then is photographed (Fig. 13B). Respiratory sounds cannot be heard on the expanded side.

Special roentgenograms during inspiration and expiration are desirable for the demonstration of every single case, because the normal lung is deficient in air during expiration and this could be considered as an atelectasis (Fig. 17). The state of deep inspiration shows the normal condition. The two roentgenograms together are of greatest importance in establishing the diagnosis and in demonstrating the condition (Fig. 112).

**Valvular Emphysema with Partial Mediastinal Shift.** Valvular stenosis occurs not only in a main bronchus but also in one or two lobar bronchi. In such a case chiefly the normal lobe of the involved side is compressed during expiration as the result of the inflation of the affected lobe. The affected lobe is emphysematous and the normal one is deficient in air (Fig. 14). Naturally, in these cases the moving of the mediastinum is less than when the whole lung is involved. The case on page 47 is of this type.

<sup>\*</sup> Paradoxical movement of the diaphragm appears also in two other conditions: *pneumothorax* and *phrenic paralysis*. In *pneumothorax* the mechanism is exactly like that of valvular emphysema, even though in *pneumothorax* the air is trapped in the chest cavity and in valvular emphysema it is trapped in the "lung tree." In cases of unilateral phrenic paralysis the paralyzed leaf of the diaphragm ascends to a higher level into the chest cavity; in inspiration it ascends even higher because the sucking effect chiefly of the healthy side pulls it up. However, there is this characteristic difference between valvular emphysema and phrenic paralysis. The sucking of the healthy side which pulls the diaphragm up also affects the mediastinum and heart and in inspiration pulls them to the uninvolved side; the paralyzed diaphragm gives room for this action. Thus, in phrenic paralysis the movement of the diaphragm is paradoxical but the mediastinum shifts in inspiration to the uninvolved side in contrast to the shift in valvular emphysema (Holtzknacht's sign) to the involved side.

Pathologic paradox motion is facilitated by the physiologic paradox motion in all three conditions—valvular emphysema, *pneumothorax* and *phrenic paralysis* (Chap. 2).

CASE 2 A boy three years of age had a poor appetite for 7 months and was losing weight. Two months before hospital investigation a right lateral segment shadow could be observed (epituberculosis) (Fig. 14A) probably a rupture of a lymph node into the lateral segmental bronchus.

A few days before investigation this shadow had been moving upward (Fig. 14B and C). An obstructive emphysema developed in the right middle and lower lobes with Holtzknacht's sign to the right. The motion extended chiefly to the heart (Fig. 14B and D). A new rupture into the stem bronchus was evident.





FIG 15 Anteroposterior x rays of a nine month-old infant A Film showing valvular emphysema of the whole left lung and of the right middle and lower lobes due to rupture of tuberculous lymph nodes into the left main bronchus and into the right stem bronchus B Film taken after the ruptured content was removed from the bronchi

A bronchoscopic examination revealed the lumen of the stem bronchus to be blocked by pale granulations. After these had been removed a caseous mass the size of a small hazelnut was discharged through the fistula on the medial wall into the bronchus. It was also removed.

The obstructive emphysema ceased immediately and the Holzknacht sign, 10 days later.

The lateral segmental atelectasis probably was caused by the rupture of a tuberculous lymph node, the same process produced the obstructive emphysema of the middle and lower lobes. Therefore in this case it was observed that an atelectasis and, simultaneously, a valvular emphysema were caused by ruptures of tuberculous lymph nodes.

In this case valvular stenosis was in the right stem bronchus. The right middle and lower lobes were excluded from respiration. The more strongly moving part of the mediastinum corresponding to the heart, lay between the affected lobes of the right lung and the normal left lung (Figs 14 and 110).

Valvular emphysema may occur on both sides at the same time (Figs 15 and 113). Thus also happened in Case 10, described in Chapter 12. The evidence in these cases, at variance with the original statement of Holzknacht,<sup>21</sup> is that a motion of the mediastinum is not caused in every case by a unilateral stenosis. In bilateral valvular emphysema the middle shadow moves during inspiration in the direction of the side where the greater part of the lung is affected (Fig 16).

In the cases shown in Figures 15 and 113 the valvular emphysema extended over the whole left lung and the middle and lower lobes of the right lung. In these cases only that part of the middle shadow moved

## ROENTGENOLOGY

which lay between the normal upper lobe of the right lung and the affected left lung that is the part above the heart

As stated previously in bilateral or unilateral stenosis in which the whole lung is not affected—that is in which the main bronchus is not stenosed—that part of the middle shadow which lies between the normal and the affected part of the lungs performs the motion or the greater motion (Fig 16) This is the *partial* movement of the mediastinum and heart

*Irregular Transitory Valvular Emphysema* Complete valvular action occurs when the air is completely trapped in expiration and the absorbed portion continually replaced in inspiration However in some cases of simple stenosis (bypass valve mechanism) the mediastinum and heart happen to shift in expiration to the healthy side This phenomenon is due to occasional valvular action of the stenosis which action is an *irregular transitory* form between simple stenosis and valvular emphysema

*Incomplete Valvular Emphysema* In incomplete valvular emphysema which is a regular transitory form the bronchus is in a blocked state during expiration only so long as the pressure of the outward going air lasts It is open during inspiration and in resting position the valvular action is incomplete therefore the lung is not inflated During expiration which follows the deep inspiration the bronchus is blocked the air cannot pass out and the lung remains in a state of deep inspiration In the narrowing

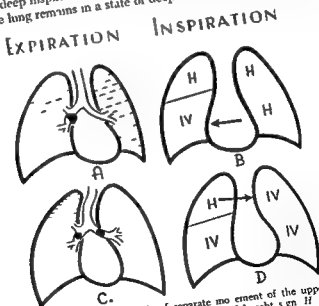


FIG 16 Schema explaining the rule of separate movement of the upper and lower portion of the mediastinum The arrows show the Holzknöchel sign H healthy IV normal



### EXPIRATION RESTING POSITION INSPIRATION



FIG 17 Anteroposterior x rays of an eight year old girl. Incomplete valvular emphysema is observed in the left lung. A Film taken on expiration. B Film taken on inspiration. Tuberculous lymph node ruptured into the left main bronchus. Note the swing of the middle shadow to the right side on expiration and to the left on inspiration. C Schema of phases of respiration in a case of incomplete valvular emphysema. The large arrow shows the Holzknacht sign.

thorax there is not enough room for the lung in its normal position therefore it pushes the mediastinum and the heart over to the uninvolved side and the diaphragm toward the abdominal cavity (Fig 17A). During the pause in breathing which follows the expiration the obstructed bronchus opens and air passes out and the organs resume their normal positions. But the stenosis still exists therefore in accordance with the principle of the Holzknacht sign the middle shadow shifts during inspiration to the involved side just as in a simple stenosis (Fig 17B).

In simple stenosis the mediastinum and heart perform the half movement of a pendulum to the involved side on inspiration (Fig 11) in an incomplete valvular emphysema the movement is like a pendulum—on expiration to the normal on inspiration toward the involved side (Fig 17A B and C) and in complete valvular emphysema on expiration to the uninvolved side (Fig 13).

Valvular emphysemas are demonstrated in Figures 13-17 20 46 60 65 110 112 and 113.

### Atelectasis

The term *atelectasis* in pathology means airlessness of a lung. Translated literally it signifies *incomplete dilatation* (from *ateles* meaning *insufficient incomplete, imperfect* and *ektasis*, meaning *dilatation*). This originally meant the incomplete expansion of a lung at birth. From this use the term has been borrowed and accepted—incorrectly, because in connection with bronchial disease atelectasis occurs when the bronchus is completely blocked and the air completely absorbed from the alveoli. However, the author has often observed roentgenographic densities which were considered atelectasis but the involved pulmonary tissues were certainly not completely airless. This happened as follows:

1. It occurred in cases of "milk glass shadow" of an entire lung because the air which lightens the density must be present in the "atelectatic" lung itself (Fig. 18). It cannot be before or behind the airless tissues as it is supposed to be in cases of lobar "milk glass shadows."

2. This may occur in cases of so called "compressive atelectasis" because it is a well known fact that air cannot be squeezed completely out of the alveoli by compression.

3. A slight amount of air may remain in the alveoli even if because of the weakened respiration most of the air is absorbed.

4. In several cases of atelectasis the author heard the wheezing sound of air occasionally passing in during inspiration.

From clinical work the author<sup>22</sup> believes that the original meaning of the term *atelectasis* (the *incomplete expansion or diminished air content*)



FIG. 18. Anteroposterior and lateral films of a sixteen-month-old boy who aspirated a piece of vegetable 5 to 11 days before his admission to the hospital. A. Film showing "milk glass shadow" of the right lung with displacement of the mediastinum and heart before bronchoscopy. B. Film taken a few hours after removal of the foreign body showing the same displacement but less density of the left lung. The lung before removal of the foreign body (film A) certainly contains a small amount of air and a few hours after it contains more (film B). In cases in which the patient's history is not known both densities would be considered atelectasis.



is more accurate and more expressive than the usual definition (*complete airlessness*). Generally it may be said only that atelectasis due to obstruction compression contraction or weakening of respiration appears *clinically* when less air enters the lung than is absorbed. On the other hand it is very clear that valvular emphysema appears when more air enters the alveoli than is absorbed. Naturally there are many cases in which the air completely disappears from the alveoli. This chiefly happens when the delicate bronchi and bronchioles are blocked by secretions and air entering the major bronchi cannot penetrate the alveoli. The shadow of such a lung is very dense and massive (Fig 108).

There are two distinct forms of atelectasis—collapse of the lung and drowned lung.

**Collapse of the Lung.** When the lung collapses the bronchus is suddenly occluded and air is quickly absorbed from the blocked part of the lung. Consequently there is considerable decrease in volume with total or partial collapse of the lung (Figs 18 64 66 81 110 and 116).

A collapse of the lung arises with absolute certainty only in a portion of the lung covered by pleura (in the whole lung or in a lobe). The occurrence of a purely segmental atelectasis is considered doubtful. It has been noted by thorough examination that a collateral change of air is possible in partial obstruction in which pulmonary tissue composing less than a lobe is involved.

**Drowned Lung.** The drowned lung occurs if the lung is slowly and gradually blocked. The inspiratory movements suck the transudate and/or



A

B

FIG. 19. Anteroposterior x-ray of a thirty-five-year-old woman. The left lung atelectatic because of a lesion in the left main bronchus. The mediastinum and the heart shift more to the left side in inspiration. A. Film taken on expiration and B. on inspiration; note the Holzknecht sign.

the exudate into the blocked part of the lung. The drowned lung does not decrease to as great an extent as a collapsed one (Fig. 108).

In both forms of atelectasis the mediastinum and the heart are placed on the involved side and shift more toward the same side in inspiration (Holzknecht's sign). The mediastinal organs follow the expanding thoracic wall together with the atelectatic lung on inspiration. By this sign one is able to differentiate between valvular emphysema and atelectasis (Figs. 13, 17, 19).

If a portion of the lung is atelectatic even for a single day, it does not clear up in every case immediately after the removal of the foreign body. Generally, 1 to 3 days is required for the alveoli gradually to fill with air. Only then do the collapse and the Holzknecht sign disappear from the affected lung. One should not be misled by the Holzknecht sign in these cases. A movement of the mediastinum may be observed in a clarifying lung for 2 to 3 days and also after the complete removal of the foreign body.

### *Importance of the Shift of the X-ray Middle Shadow*

In the foregoing discussion of the problems of the x-ray signs of the pathologic respiratory mechanisms it was seen that independent of the degree of the stenosis the middle shadow shifts during inspiration always toward the involved side. By "independent of the degree of the stenosis" we do not mean of course that even the slightest bronchostenosis produces a motion of the mediastinum, but that we perceive the Holzknecht sign in cases of simple but substantial stenosis as well as in cases of valvular emphysema and atelectasis.

The observation is based upon the dynamics of respiration and upon the consequent volume alteration. Therefore attention has to be turned to the whole process of respiration, since one cannot judge from a single glance. Above all attention should be paid to the movement of the middle shadow and of the diaphragm.

Experience shows that not only the laryngologist but also most of the roentgenologists do not give sufficient attention to the pathologic movement of the mediastinum and the heart which constitutes a very important x-ray sign. Very frequently nonopaque foreign bodies, perforation of the tuberculous lymph nodes, tumors and many other bronchial or pulmonary diseases can be localized or even discovered only by this sign. Observation of the behavior of the x-ray middle shadow is as important in the fluoroscopic routine as is the examination of the lung fields, retrocardiac area or movements of the diaphragm. During this examination the patient must remain motionless and face the examiner directly. Agitated children and infants must be held firmly. The diaphragm of the x-ray apparatus

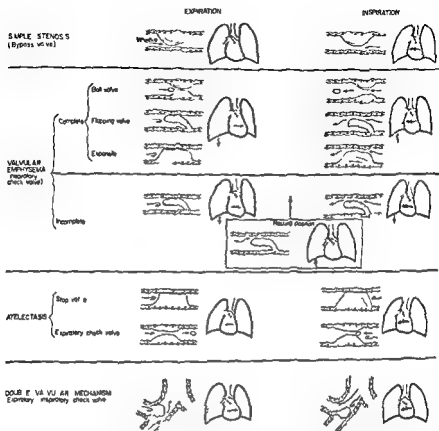


FIG. 20 Schematic drawing of the pathologic respiratory mechanisms accompanying bronchial obstruction correlating the types of obstruction with the corresponding

is narrowed so that only a vertical thin strip is seen with narrow lung fields on both sides of the middle shadow. Thus besides the inspiratory narrowing the slightest shift is observable. In infants and young children, particular attention should be paid to the expiratory widening and inspiratory narrowing of the mediastinum. In crying infants the long expiration normally decreases air content of the lungs to such a degree that the mediastinum seems to reach the lateral thoracic walls on both sides.

Pathologic respiratory mechanisms associated with bronchial obstruction and their corresponding x-ray signs are demonstrated in Figure 20.

## X RAY APPEARANCE OF THE PULMONARY UNITS

*Segmentology* is a branch of modern pneumology. Some decades ago only the lobes (and the lobuli) were considered units of the lung. Now the segment also a portion of the lobe is accepted as a pulmonary unit (Fig 21). The segment is a roughly pyramid shaped bronchovascular unit isolated from the others by a layer of connective tissue. All the segments have a pedicle consisting of a major bronchus (segmental bronchus) and an artery (segmental artery). The bronchus and artery run close to each other; their names also correspond. The veins are located in the intersegmental septums. The surgeon performing segmental resection follows the veins when isolating the intersegmental connective tissue.

Several pulmonary diseases (Asthma, focus, abscesses, etc.) appear in certain segments. Others (atelectasis or pneumonia) due to many different

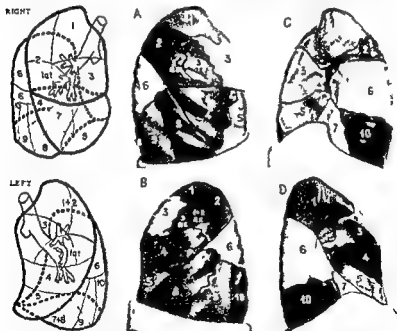


FIG. 21. The author's model of segments of the lungs. A, B, Lateral views of the right and left lungs. C, D, Medial views of the right and left lungs. 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, subsegment.

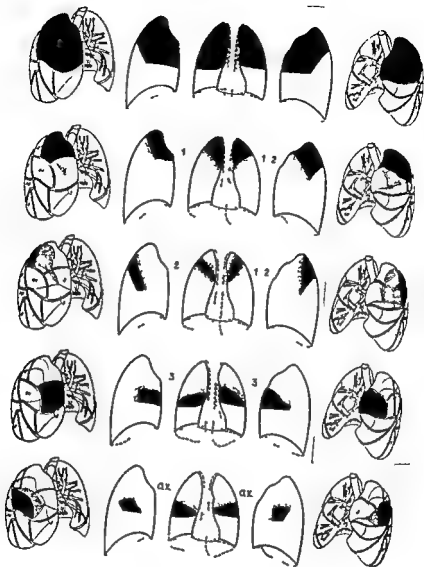


FIG. 22 X-ray guide to the shadows of the right upper lobe and the left truncated lobe and their segments. In the center are sketches of the segments from the

by Kovács, Jr. 1943) in three dimensions (Three dimensional schematic drawings)

causes epituberculosis etc.) occupy one or more whole segments. Physiology roentgenology and pathology of a segment differ from those of larger pulmonary units (lungs or lobes). Therefore the author uses the term *segmentology* to include not only the segmental anatomy but also the physiology roentgenology and pathology of the pulmonary segments. The differences are discussed in this chapter and Chapters 7 to 12.

The bronchologist should understand the x-ray appearance of the pulmonary segments because the cause of a segmental shadow is frequently located in the segmental bronchus. Thus in segmental atelectasis the supplying bronchus must be examined with precision.

Atelectasis of a lobe and a segment differ in many respects. A lobe covered with intact pleura is able to collapse considerably. The interlobar margin of the shadow of an atelectatic lobe fixed by adhesion to the chest wall is concave because of the diminished volume.<sup>6</sup> In segmental atelectasis neither collapse which should not be confused with a fibrotic shrinkage nor concavity of the segmental border is observed. One reason for this is that the intersegmental fixation keeps the segment stretched; the

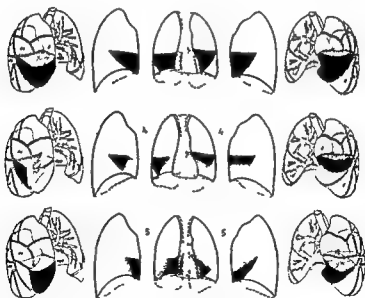


FIG. 23. Schematic drawings of the x-ray shadows of the segments of the right middle lobe and lingula as explained in Fig. 22. (Three-dimensional schematic drawings by Koudits, Jr.<sup>10</sup>)

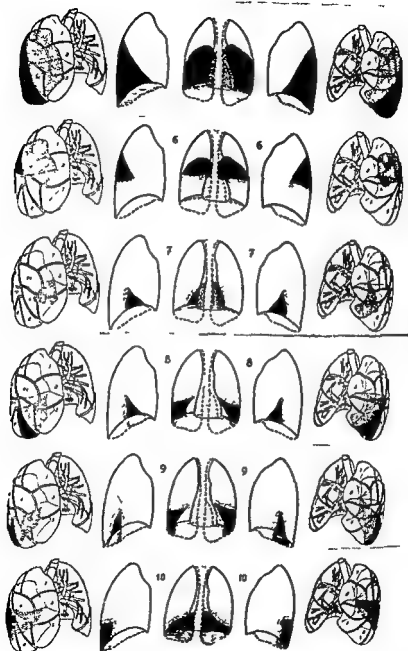


FIG. 24 Schematic drawings of the x-ray shadows of the segments of the lower lobes as explained in Fig. 22 (*Three-dimensional schematic drawings by Kovács Jr.<sup>14</sup>*)

other is that segmental atelectasis rarely appears as a complete airlessness for retained bronchial secretions transudate or exudate are present. For the roentgenographic appearance of the pulmonary segments see Figures 22 to 24.

Several segments are larger than can be seen on roentgen films because the segments extend into and are covered by shadows of the mediastinum (on anteroposterior film) and diaphragm (on both anteroposterior and lateral). These hidden portions of the segments are shown in Figures 22 to 24.

The intersegmental borders of the segmental shadow are dotted in Figures 22 to 24 to show that these borders in contrast with the interlobar fissures rarely appear pronounced and distinct but rather indistinct, uneven or zigzag (Figs 69-74, 93-95, 97, 98, and 111). One explanation of this phenomenon is the fact that the intersegmental septums are not plain and smooth as the pleural surfaces of the lobes but uneven or bumpy like basalt rocks. In these intersegmental surfaces the pentagon-shaped lobuli of the neighboring segments fit each other as a mold and cast and thus lobuli of the healthy segments protrude into the atelectatic ones (Fig. 25).<sup>11</sup>

The other reason for this phenomenon is the so-called "collateral ventilation" which may permit air to enter the atelectatic segment near the border through the pores between the alveoli of the neighboring segments making these surfaces indistinct or fuzzy.



FIG. 75 Photograph of a right upper lobe. The segments (numbered) are filled with differently colored latex and after solidification separated in the intersegmental septums. A Lateral view of the lobe. B The intersegmental surfaces of the segments which are not smooth but uneven and angulated like basalt rocks. This finding explains the phenomenon that in a ray films the borders of a segmental atelectasis appear indistinct or zigzag because the healthy segments protrude into the atelectatic ones axillary.



Appearance of the different pulmonary units in x-ray films are shown in the following figures

#### RIGHT PULMONARY UNITS

*Lung*, 103

*Lobes*

Upper, 37, 79, 81 93, 109

Upper and middle, 45

Middle, 48 49, 67, 96, 110

Middle and lower, 63, 65, 91, 115

Lower, 61, 63, 66 116

*Segments*

Posterior, 95

Anterior, 69, 94

Axillary, 70

Lateral, 14 71

Medial, 62, 72

Apical 54 73, 80

Medial basal, 88

Anterior basal, 74

Lateral basal, 97, 111

Posterior basal 98

#### LEFT PULMONARY UNITS

*Lung*, 18, 19, 64, 83, 103

*Lobes*

Upper, 51, 90, 99

Truncated, 75

Lingula, 76, 102

Lower, 44, 46

*Segments*

Posterior, 100

Anterior, 101

Axillary, 88

Superior lingular, 53

Inferior lingular, 50

Apical, 47, 56

Posterior basal, 55, 89

Lateral and posterior basal 77

#### Summary

The mediastinal flutter resembles the action of a pair of scales (Fig 11)

In the dynamic process of a pathologic respiratory mechanism, the changes of volume are of primary importance. The whole process of respiration must be observed. Attention must be focused chiefly on the motion of the middle shadow and of the diaphragm.

In pathologic respiratory mechanisms the middle shadow shifts during inspiration—independently of the degree of the stenosis—toward the involved side.

A movement of the mediastinum can be produced not only by a unilateral constriction, but also when the middle shadow shifts in bilateral stenosis during inspiration toward the side where a greater part of the lung is affected. This is at variance with the statement of Holzknecht on the subject.

In bilateral or unilateral stenosis (but only in stenosis not affecting the total lung—hence, where the constriction does not affect the main bronchus) the motion or the greater part thereof is performed by that part

of the middle shadow which lies between the affected and normal parts of the lungs

Segments are now considered pulmonary units along with the lobes. Segmental anatomy, physiology, roentgenology and pathology differ in several respects from those of larger pulmonary units (lungs and lobes).

Atelectasis of a segment differs from that of a lobe or lung. The most important differences are (1) An atelectatic segment almost never collapses as well as an atelectatic lobe or lung. (2) Although its appearance of the interlobar borders is marked and distinct like that of a lobe, the intersegmental borders are rather indistinct, uneven or zigzag. Classification of mechanisms of all pathologic respiratory conditions and corresponding x-ray signs are shown in Figure 20.



# Instruments, Techniques, and Indications

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## INSTRUMENTS

The bronchologist's instruments are divided into five groups (1) the bronchoscope (an endoscope or tube with lighting apparatus) (2) magnifying instruments (3) auxiliary instruments (forceps probes hooks curet aspirating and irrigating tubes sponge carriers bougies catheters injecting needles snares knives scissors electrodes diathermic coagulators magnets etc.) (4) photoapparatus and (5) operating room equipment

### *The Bronchoscope*

Two types of bronchoscopes exist one with proximal and the other with distal lighting. Both types are satisfactory for use in both diagnosis and therapy. However the proximal type has some advantages in bronchoscopic surgery the advantages of the distal system are evident in diagnostic procedures.

For certain special procedures a bronchologist may use special tubes (infant costophrenic split etc.)

**Proximal Type** The light source of the proximally lighted bronchoscope is attached to the ocular end of the tube. This light system is composed of a lamp a lens and a reflecting mirror. The light system of earlier proximally lighted bronchoscopes<sup>1,23</sup> (Fig. 26) was fixed above the ocular end of the tube therefore the strongest light appeared on the ring of the tube and caused a disturbing reflection even when the ring was painted black. The light of the newer bronchoscopes<sup>4,5,6</sup> (Fig. 27) is hidden inside the tube and there is no reflection.

**Distal Type** The lamp of the distally lighted bronchoscope is placed on the inner aspect of the distal lip of the tube<sup>7,8</sup> (Fig. 27). Bronchoscopes of this type other than the one shown in Figure 27 differ slightly

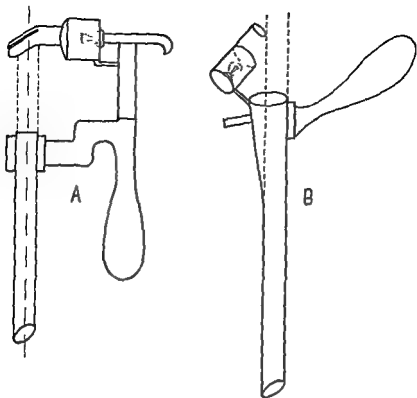


FIG 26 Schematic drawing of the bronchoscopes of (A) Brunings<sup>14</sup> and (B) Hallinger<sup>8</sup>

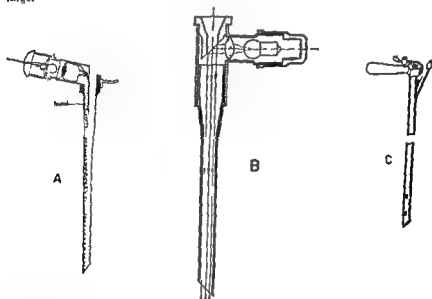


FIG 27 Schematic drawing of the bronchoscopes of (A) Kassay,<sup>29</sup> (B) Kallav<sup>13</sup> and (C) Jackson<sup>32</sup>

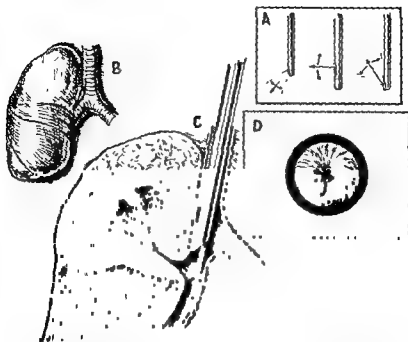


FIG 28 Drawings of telescopic examination A The distal end of the Foroblique, right-angle, and retrograde telescopes B A malignant growth in the right upper lobe C A bronchoscope inserted into the bronchus D A magnified view of the malignant growth

Makers, Inc., New York }

### Magnifying Instruments

For magnification the bronchologist uses double-lens systems and telescopes

**Double-lens Systems.** These systems may be focused and are attached to the ocular end of the tube

**Telescopes.** The telescopes are inserted through the standard bronchoscopes There are forward, Foroblique, right-angle, and retrograde telescopes, the proper one is selected according to the angle of bronchial branching (Fig 28)

### Auxiliary Instruments

Among the auxiliary instruments are bronchoscopic forceps. Two types of bronchoscopic forceps are used alligator and cannulated (Fig 29).

Additional and frequently used auxiliary instruments are shown in Figure 30

**Alligator Forceps** Alligator cup-bite forceps are used for biopsy, and alligator grasping forceps are used for the removal of flat-shaped foreign bodies

**Cannulated Forceps.** Many types of cannulated forceps are in use (grasping, peanut, rotating, screw, nail, pin bending, button removal, four bladed, cylindrical, approximation, costophrenic, dilating, tack pin, safety pin, upper lobe, etc.) Also, a flexible forceps may be combined with a telescope for biopsy or foreign body removal with good visualization of bronchi running at a right angle to the main or lower lobe bronchus

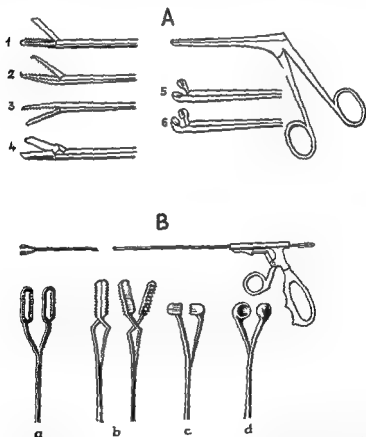


FIG. 29 Two types of bronchoscopic forceps A Alligator type B Cannulated type Both may be equipped with different kinds of blades 1 to 3, various kinds of grasping forceps, 4 bronchoscopic scissors, 5, straight forceps, and 6 angular cupped forceps, a, fenestrated peanut forceps, b, dilating forceps, c, side-curved grasping forceps, d, biting forceps

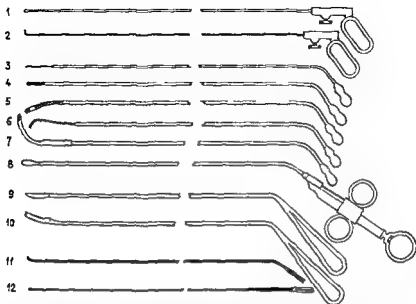


FIG. 30 Commonly used bronchoscopic instruments 1 probe 2 hook 3 injection needle 4 aspirating tube with serrated end (the rubber tube does not slide off) 5 flexible aspirating tube 6 and 7 instruments for aimed bronchiography 8 snare 9 and 10 bronchoscopic knives 11 cautery electrode 12 diathermic electrode

### *Photoapparatus*

For bronchoscopic photography Holinger<sup>22</sup> used a proximal lighting system. In the instruments of Dubois<sup>23</sup> and Jackson<sup>24</sup> a light is conducted into the bronchi by a quartz rod and the photographs are made through a telescope<sup>25</sup>.

### *Operating room Equipment*

In operating room equipment the Jackson bronchoscopic table<sup>26</sup> and the C. L. Jackson mobile bronchoscopic units<sup>26</sup> are especially useful.

## TECHNIQUES AND APPROACHES

### *Premedication and Anesthesia*

Bronchoscopic procedure is a minor operation which can be performed on outpatients as well as inpatients. However, some preparation is indicated. The patient generally is referred to the bronchologist by a hospital or by a physician and has been examined at least with respect to lung and heart. Anteroposterior and lateral x-ray films of the thorax must be made



The result of these and other examinations must be available to the bronchologist who completes them if required. The patient should be examined to exclude organic diseases (cardiac lesion hypertension acidosis diabetes etc.) Without some knowledge of the patient's physical condition and of any lesion of the chest organs to be examined bronchoscopy would be useless or dangerous.

The forenoon is the favorable time for bronchoscopy when the patient and the doctor are well rested. On the day of the examination the patient is not allowed food or water for 5 hours prior to bronchoscopy and of course a clean mouth is necessary.

The patient experiencing bronchoscopy for the first time is generally nervous and requires some psychologic preparation. Chevalier Jackson<sup>1</sup> calls this a "sermon on relaxation or vocal anesthesia." In other words the patient should be told about bronchoscopic procedure and its minor inconveniences and along with this he should be instructed on how to relax so as to be helpful and cooperative. This is a very important part in the patient's preparation for bronchoscopy.

There is a variety of opinion with regard to anesthesia for bronchoscopy. One group of bronchologists favor local anesthesia.<sup>23</sup> The procedure presents fewer hazards than that for general anesthesia and the patient can cooperate if necessary. Others prefer complete narcosis unless there are positive contraindications that make it undesirable or unsafe. Most bronchologists use neither local nor general anesthesia for bronchoscopy in infants and young children. Others<sup>24</sup> believe that restraint by "mummification" (no anesthesia) is inhuman and unnecessary.

To avoid the role of moderator between the controversial groups an impartial description of the different methods follows.

**Local Anesthesia** For premedication prior to local anesthesia pentobarbital sodium (Nembutal) and meperidine hydrochloride (Demerol hydrochloride) are generally used as sedatives and morphine and scopolamine as opiates. In addition many bronchologists use atropine for diminution of the bronchial secretion; however some are inclined to omit atropine since bronchoscopy is often performed to obtain secretions for bacteriologic and cytologic examination and atropine decreases this possibility.<sup>25</sup>

Premedication must be given at least 1 hour prior to the examination. Not analgesic but soporific and antispasmodic effects are desired. Preoperative medication depends on the age and general condition of the patient. None is used in old or debilitated individuals or those with known respiratory obstruction.

The bronchologist who prefers local anesthesia uses general anesthesia only when anatomic difficulties are presented and when the patient is uncooperative or mentally unfit. This group of bronchologists as a rule

use no topical or general anesthetic in infants or children but adequate sedation is administered unless contraindicated by respiratory obstruction<sup>28</sup>

The drugs generally used in local anesthesia are cocaine and tetracaine (Pontocaine hydrochloride). These are given in various solutions of cocaine in 1 per cent (10 mg in 1 ml) or in 4 per cent (40 mg in 1 ml) and Pontocaine in 0.5 per cent (5 mg in 1 ml) with considerable satisfaction.<sup>29</sup> There have been some toxic effects from the use of 10 per cent cocaine (100 mg in 1 ml) or 2 per cent Pontocaine (20 mg in 1 ml) but no serious toxic effects were observed when the weaker solutions mentioned above were used. The newer drugs Articaine hydrochloride and Cyclaine hydrochloride are less toxic than Pontocaine and cocaine.

Local anesthetic procedure in the Jackson clinic consists of spraying lightly with 2 per cent Pontocaine or 2 per cent Articaine in patients with bronchial asthma or others suspected of having drug sensitivities (20 mg in 1 ml for both drugs). The spray is directed first on the tongue palate and posterior pharyngeal wall then directed toward the larynx as the tongue is held forward and the atomizer tipped downward. Three to five minutes are allowed to elapse between spraying and instillation for toxicity is based not only on total dosage but also on the rate of absorption. Then 1 ml of 10 per cent (100 mg) cocaine is instilled a few drops at a time with a laryngeal syringe under mirror guidance. Then 6 or 8 ml (60 or 80 mg) of Articaine is instilled 2 ml at a time after each instillation the patient is instructed to incline his body to one side and then to the other side so that the solution gravitates into both sides of the bronchial tree.

The author favors local anesthesia and uses the above described method with some modifications. Pontocaine and cocaine are not administered simultaneously but alone in each case. Pontocaine is used routinely however in cases of bronchial asthma cocaine is used since Pontocaine is particularly toxic in patients with bronchial asthma and allergies. Before spraying and instillation every patient gargles for 1 minute with 4 ml of 0.25 per cent (1 mg) Pontocaine or with 0.5 per cent (2 mg) cocaine to test his sensitivity.<sup>4</sup> If within 5 minutes no toxic effects such as psychic excitement pallor perspiration tachycardia tachypnea pupil dilatation urticaria glottal edema vertigo nausea or malaise are observed the procedure is continued. While this is not an altogether precise and reliable test for sensitivity for want of a better and simpler method this procedure is used and seems to yield some protection for both bronchologist and patient.

For spraying and instillation the amount of 0.5 per cent Pontocaine used does not exceed 10 ml (50 mg) and the amount of 1 per cent cocaine (150 mg) does not exceed 15 ml. However the total amount of the solu-

tions are not used in most cases, when the descending solution no longer causes coughing, instillation is discontinued. On the average, 7 to 8 ml Pontocaine (about 40 mg) and 0 to 11 ml cocaine (about 100 mg) are used. Not only are these drugs less toxic in smaller amounts and lower concentrations, but also the larger amount of fluid supports deeper penetration and a more uniform distribution of the anesthetic in the bronchial tree. To retard absorption, the spraying and instillation time is extended as long as 10 minutes.

**General Anesthesia.** Bronchologists<sup>22</sup> who prefer general anesthesia use both topical and general anesthetics unless respiratory obstruction, cardiac conditions, or other contraindications are present. In children, premedication and the open ("open drop") method of administering ether are generally used. In adults, premedication, curare or succinylcholine chloride as relaxants, and Pentothal Sodium given intravenously are used as general anesthetic; these may be continued with nitrous oxide and oxygen or other agents.

*Patients scheduled for general anesthesia should be hospitalized. Outpatients require an attendant if local anesthesia has been used and a sedative has been given because it is extremely dangerous to discharge a patient alone while he is under soporific effect.*

#### *Position of the Patient and Introduction of the Bronchoscope*

The endoscopic operating room must be dimly lighted, because bright illumination disturbs the bronchoscopic view. However, it must be sufficiently lighted to allow control of the patient and the instruments.

In the early days of bronchoscopy the procedure was performed in the sitting position. In Europe this position is still used, especially by German specialists,<sup>23</sup> and lately it has been proposed even in America<sup>24</sup> in spite of the fact that for decades the generally accepted practice has been to place the patient in the dorsally recumbent position.<sup>4</sup> The recumbent position is more comfortable for both the patient and bronchologist. The patient lying on a well padded flat table, can relax more easily and be more readily controlled; the bronchologist achieves more efficient teamwork and a smoother and better technique.

For bronchoscopy the centers of the patient's scapulae rest on the edge of the table. The head must be free to move and ready to receive support by an assistant or by a mechanical head rest attached to the operating table. To introduce the rigid bronchoscope it is necessary to elevate the head from 10 to 15 cm above the level of the table (Fig. 31). In this way the angles formed by the oral cavity, pharynx, and axis of the trachea can be straightened. As the bronchoscope is advanced, the position of the head must correspond to the axis of the bronchus to be examined, i.e., if the left upper lobe bronchus is being inspected, the head must be turned and

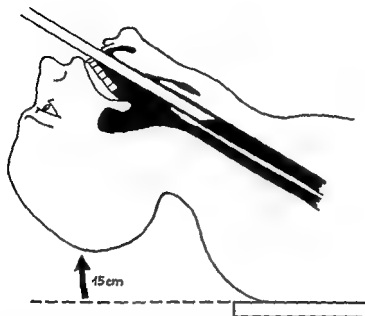


FIG. 31 Proper position of the patient's head for introducing a bronchoscope

displaced far to the right. If the right middle lobe bronchus is being explored the head requires slight displacement to the left and considerable lowering. If the examiner tries to explore the bronchi only by lateral movement of the proximal end of the tube without simultaneous movement of the head the rigid and hard tube will injure the tongue, teeth, gingiva or larynx. The pain compels the patient to make defensive movements and this small error can defeat the examination of a sensitive patient.

Unless protection is provided the bronchoscopic tube will rest on the patient's upper teeth and exert pressure. The bronchologist may protect the teeth by laying gauze over them and relieve pressure with the fingers of the left hand by allowing the tube to lie and slide on the fingers. It is of little importance if the doctor feels some discomfort on his fingers but the patient must not suffer any discomfort from pressure on his teeth. The bronchologist exposes the glottis by introducing the tube into the patient's mouth along the right side of the tongue and by elevation of the epiglottis and all the tissues attached to the hyoid bone. The trachea which is entered through the exposed glottis is the second subject of the bronchoscopic examination.

In searching for a lesion in the bronchial tree or lungs the supposedly uninvolved side is first viewed. Naturally every bronchus must be in

## CLINICAL APPLICATIONS OF BRONCHOLOGY

spected step by step the most precise search being carried out on those that lead to the lesion as evidenced by previous examination (percussion auscultation pectoriloquy roentgenogram etc) The spurs the wall and the mucosa of the bronchi must be viewed with precision their walls should be examined with a probe in magnified image secretions for bacteriologic or cytologic study and a biopsy specimen for histologic examination should be taken During the examination the bronchologist may ask the patient to cough by this action secretion may be brought up to the orifice of one or another bronchus and the involvement of a certain portion of the lung can be identified Also physiologic motions of the bronchi are always under observation All the bronchi cannot be examined in every case with such precision if one tries the prolonged procedure may cause unnecessary discomfort to the patient The bronchoscopic examination must always be exact but as short as possible Bronchoscopic views are shown in Figure 32

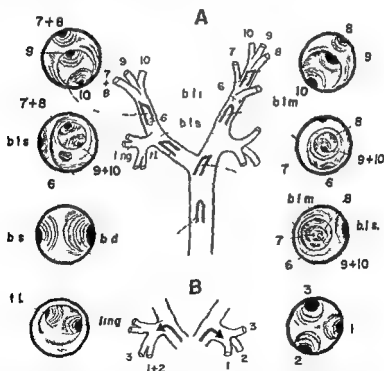


FIG 32 Bronchoscopic guide The patient is in dorsal recumbent position. A The various positions of a bronchoscope during bronchoscopic examination and the corresponding views B The telescopic view b1s the upper lobe bronchus b1m the middle lobe bronchus b1l the lower lobe bronchus b2s right bronchus b2l left bronchus t1 truncated lobe bronchus ling lingular bronchus The segmental bronchi are numbered

### *Procedures for Infants and Young Children*

Bronchoscopic procedures are no more hazardous in infants and in young children than in adults. However the delicate vulnerable tissues may be slightly contused this must not be ignored. Previous mucosal or parenchymal inflammation, dyspnea and exhaustion in the fight for air increase the general reaction. Nevertheless shock in its well known surgical form never follows a cautiously performed and time-limited bronchoscopy. In the hand of a skilled bronchologist a single careful insertion of the tube never causes marked trauma and never inflicts surgical shock. However insertion repeated three or four times and prolonged use of the bronchoscope may injure the larynx. Therefore during a bronchoscopic examination the bronchologist should endeavor to avoid repeated introduction of the tube and more important the duration of bronchoscopy in infants and young children must be as short as possible. As a rule the time of the procedure is measured and limited according to the age of the patient; in infants the time limit is 10 minutes.

For a bronchologist to be unable to discontinue bronchoscopy is the greatest error. In case of the presence of a foreign body failure is undoubtedly a very disappointing experience but the experience is a great deal more unpleasant if often repeated or prolonged bronchoscopy contributes to the death of a child. One cannot comfort oneself with the argument that not he but the foreign body was responsible for a child's death. Every bronchologist is obliged to know the rule. In children especially in infants and very young children do not prolong or repeat bronchoscopy at close intervals even in case of failure stop and postpone further bronchoscopy.

However in infants and children the organism tends to recuperate more quickly than in adults. Disease injury or contusion in a child heals easily and quickly. Local bronchoscopic treatment is more suitable for pulmonary lesions (bronchiectasis abscess etc.) in children than for these diseases in adults.

The tissues of an infant or child are loose and vascular and swell easily thus they are predisposed to edema. A child's tissues contain much lymphoid tissue. These statements refer particularly to the larynx and respiratory tracts. The larynx especially has a peculiarly loose structure in infants. As is well known the congenital inspiratory stridor is one of the results of this looseness (Extremely loose mucosa of aryepiglottic folds and arytenoid cartilages sometimes permits the epiglottis itself to be drawn into the glottis and thereby causes stridor during inspiration). These circumstances predispose the larynx of young children to edema and inflammation which may complicate bronchoscopic procedure.

The most sensitive region of the larynx is the subglottal area. Here the rigid ring of the cricoid cartilage embraces the lumen of the larynx. This

is the only point in the laryngotracheobronchial system that is unable to dilate. The cricoid cartilage in this region is covered only by a relatively thin mucosa, therefore, it is easily contused and traumatized. This is especially true in certain circumstances, such as in the use of an oversized tube when the mucosa may be jammed between the hard cartilage and the even harder metal tube. The vascular mucosa responds to the trauma with inflammatory edema. Thus, the alarming *subglottal edema* arises.

Fear of subglottal edema led writers at the beginning of the century, in the era of Killian and Brunings,<sup>14</sup> to form a rule to the effect that a skilled examiner of any child under three years of age and an unskilled beginner examining children under six years of age should perform only "inferior bronchoscopy," which meant the insertion of the tube through the stoma of a previously performed tracheotomy. These writers, as an explanation of this rule, said that through the stoma a wider and shorter tube could be used, which increased visibility and facilitated orientation and manipulation in depth. Another argument was that subglottal edema would in any case require tracheotomy on the first or second day, thus, to avoid the ensuing laryngeal stenosis with its alarming symptoms, it was more reasonable to perform a preliminary tracheotomy in preparation for "inferior bronchoscopy."

Progress of bronchoscopic technique eliminated the need for this rule, which, to the author's knowledge, was never accepted in America even in the earliest days of the Jackson school. Today, no one speaks about "inferior" or "superior" procedure, only one type of bronchoscopic procedure exists, and that is the superior type. The inferior method is useful only in an emergency. When life is in danger (e.g., when a child is suffocating because of a laryngeal or tracheal foreign body), this method may be applied, especially if a less skilled specialist is in charge.

The bronchologist dealing with infants and children is interested in the measurements of the interior of the larynx, chiefly in the diameter at the level of the cricoid cartilage. With these data he may select a tube that will easily pass through the larynx with little irritation of the mucosa and yet yield the widest view.

Naturally, in infants and children, only a delicate instrument (forceps hook, aspirating tube, etc.) may be used through the narrow tube in order to allow sufficient inspection for correct manipulation.

Not only the tissues of the larynx, but also the tissues of the trachea and bronchi are delicate and easily traumatized in infants and young children. In this area contusions or injuries may be caused by auxiliary instruments (forceps, hook, etc.) rather than by the tube. After prolonged and inexpertly performed bronchoscopy the mucosa becomes congested and produces large amounts of thick purulent secretion, which may drown the tracheobronchial tree. For this reason, in addition to the possi-

bility of subglottic edema, prolonged bronchoscopic procedure is avoided.

The tissues of children debilitated by tuberculosis, bronchial asthma, or other diseases are extremely sensitive. In such a child poorly performed or prolonged bronchoscopy may produce subglottal edema or severe mucosal inflammation more frequently and quickly than in a well-developed, well-nourished child. The bronchologist must be very careful with debilitated children and children with bronchial perforation, fistulas, or compression caused by tuberculous lymph nodes, for he is restricted to one insertion and the usual time is shortened. In treating these patients, however, he does not need a long time. Diagnosis of these conditions and the removal of ruptured granulation tissue and cheesy debris are more easily accomplished than is the diagnosis and elimination of a foreign body.

These statements are not intended to discourage the use of bronchoscopy, but rather to guard against its misuse in order to prevent its disqualification as a valuable medical procedure.

### *Bronchography and Aimed Bronchography*

Simple bronchography has been a well-known procedure for decades. In essence it is the procedure whereby opaque compounds containing iodine, in a special oily or watery fluid, are injected into the bronchi and exposed to roentgen rays from anteroposterior, lateral, and sometimes oblique directions. Roentgenograms taken in this way are called *bronchograms*.

Bronchography, especially aimed bronchography, is performed by the roentgenologist and bronchologist working as a team. Almost every team has its own technique; therefore it is almost impossible to describe a technique that is generally accepted as standard. The author uses the following method:

In a routine bronchoscopic procedure, only the major bronchi are touched by the tube and instruments, but in bronchography the contrast medium enters and irritates hundreds of minor bronchi. Therefore, bronchography requires the same premedication as, but more precise anesthesia than, bronchoscopy. Usually local anesthesia for adults and general anesthesia for infants and young children are used. Children of

bronchography than for bronchoscopy. The anesthetic solution must reach all the minor bronchi which will be exposed to the irritation of the contrast medium. An anesthetic administered to the patient in a sitting position is frequently inadequate, because the solution enters minor bronchi only in the basal parts of the lower lobe. The anesthetic penetrates only



■ small portion of the other bronchi in the upper lobe middle lobe, or lingula and of the apical segment of the lower lobe when the patient coughs and takes deep breaths. Therefore, after routine procedure has been performed with the patient in a sitting position, 2 to 3 ml from the permitted amount of solution is saved for completion of the anesthesia. The bronchographic catheter is introduced as far as the upper lobe bronchus under fluoroscopic guidance, and 1 ml of the saved solution is injected while the patient is lying in the lateral recumbent position. Then the patient turns to the dorsal and later to the ventral recumbent positions, and the catheter is advanced as far as the middle lobe bronchus. Now the rest of the solution is injected. In these positions the anesthetic solution gravitates and is inhaled by deep breaths into the bronchi that were inadequately anesthetized in the sitting position. The patient usually signalizes the incompleteness of anesthesia with a cough.

Now, the patient's bronchial tree is prepared to accept the contrast medium which will also be injected under fluoroscopic guidance. By fluoroscopic observation, a very important part of bronchography, physiologic respiratory and pulsatory movements, and caliber and length alterations of the bronchi are inspected. The normal dynamics, pathologic movements or rigidity of the bronchi should be included in a report of bronchographic examinations as well as being a part of every report of bronchoscopic examination.

With the aid of fluoroscopy, the optimal filling of the bronchial tree is controlled as the contrast medium is aspirated from major to minor bronchi by the patient's deep inspirations until the bronchi are filled adequately without alveolar penetration. Even distribution of the contrast medium can be facilitated by changing the position of the patient. First the lateral then the anteroposterior, or, if it is desirable the oblique films can be exposed. During exposure the patient remains quiet, without breathing.

Generally, it is easy to analyze a bronchogram which shows an adequately filled, healthy bronchial tree. A pathologically distorted tree may cause problems, but the real problems are the imperfect bronchograms. Usually, the bronchi of a whole lung are filled during routine bronchography. This filling may be imperfect for the following reasons:

- 1 Incomplete anesthesia

- 2 Inadequate amount of the contrast fluid. Routinely, 20 ml should be prepared for an adult bronchogram. Both smaller and larger amounts

always limits the amount of the contrast medium. The same amount will fill incompletely in one patient and cause alveolar filling in another.

- 3 Exposure too early or too late

- 4 Effective inspiration is very important in bronchography, because it

supports the contrast medium from major to minor bronchi. Extra and intrapulmonary lesions can result in inadequate sucking effect on inspiration. Extrapulmonary lesions are emphysematous barrel shaped thorax, fracture of ribs, phrenic paralysis, atrophy of muscles, pneumothorax, pleuritis, empyema, adhesions, mediastinal processes, intrapulmonary lesions are congestion, diseases with fibrosis or destruction of elastic elements, etc. In every instance of excess bronchial secretion, naturally, bronchoscopic aspiration must precede bronchography.

In instances of filling defects, a pulmonary unit—an entire lobe or segment—may remain empty or incompletely filled. Often it is particularly the affected part to be examined that fails to accept the contrast medium. This failure is seldom a result of an error of bronchography if performed by skilled hands as discussed above. The bronchus narrowed by congestion or scars or obstructed by tumor, torsion or angulation does not allow the contrast medium to enter, or the respiratory sucking effect is diminished and is insufficient to inspire the fluid into the bronchi of a pulmonary unit. In these cases special delicate tubes can be introduced into the bronchi, aimed with both bronchoscopic and fluoroscopic guidance.<sup>42</sup> Some tubes are rigid, but some are sufficiently pliable (Fig. 33A), others are flexible but hard enough for insertion into the narrowed lumen through the bronchoscope (Fig. 33B-E). With these tubes the bronchi which would remain empty on normal bronchograms can be filled. The otherwise undemonstrable cavities—abscess or tuberculous—can also be filled by this method.

Aimed bronchography may be utilized in every case of filling defect (dead tree effect) involving a pulmonary unit (lobe or segment) with positive or even negative x-ray findings. Surprising results are often obtained (Figs. 55, 58, 59, 62, 67, 79, and 80).

Naturally aimed bronchography only supplements simple bronchography; never supplants it.

Areas of indications for bronchography are extremely wide. They are discussed in detail in Chapter 13.

### *Repeated Aspiration of Bronchial Secretions and Exudates*

One of the revelations of bronchoscopy is the clinical fact that all inflammatory bronchial exudates are to a high degree cohesive and adhesive. In pre-bronchoscopic days this was not realized because these secretions, when expectorated, are diluted and lubricated with saliva in sputum. Bronchial and segmental orifices of various sizes were seen obstructed by masses of inflammatory purulent and mucopurulent exudates that could not be efficiently expelled by unaided physiologic mechanisms. Areas tributary to such plugged orifices quickly become atelectatic and hence vulnerable to infection.<sup>43</sup>

Thus in every disease in which expectoration is retarded, secretion continually accumulates in the lower airways; repeated bronchial aspirations

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- 1 Incomplete anesthesia
- 2 Inadequate amount of the contrast fluid. Routinely, 20 ml should be prepared for an adult bronchogram. Both smaller and larger amounts might cause faulty bronchograms. Smaller amounts may result in incomplete filling and larger ones, in alveolar filling. However, the optimal filling always limits the amount of the contrast medium. The same amount might fill incompletely in one patient and cause alveolar filling in another.
- 3 Exposure too early or too late
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supports the contrast medium from major to minor bronchi. Extra- and intrapulmonary lesions can result in inadequate sucking effect on inspiration. Extrapulmonary lesions are emphysematous barrel-shaped thorax, fracture of ribs, phrenic paralysis, atrophy of muscles, pneumothorax, pleuritis, empyema, adhesions, mediastinal processes. Intrapulmonary lesions are congestion, diseases with fibrosis or destruction of elastic elements, etc. In every instance of excess bronchial secretion, naturally, bronchoscopic aspiration must precede bronchography.

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Thus in every disease in which expectoration is retarded, secretion continually accumulates in the lower airways; repeated bronchial aspirations

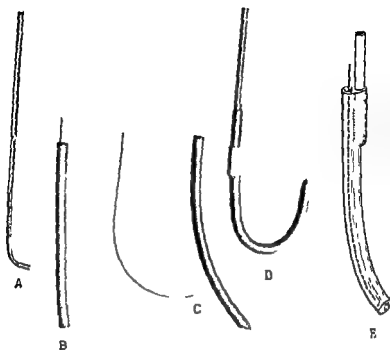


Fig  
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exam  
7, 8

and the superior segmental bronchi of the lower lobes (see also Fig. 104).  
The other type of tube has four types of changeable ends. Each of them consists  
of a spring and a rubber tubing. Three types of spring are shown

The spring keeps the rubber tubing in a straight plane. Because of this mechanism the tube is more easily directed than any other  
flexible kind, e.g. the tube with a spiral flexible end which is unsuitable for this  
purpose because of its leanness. Other kinds of tubes such as those ending in  
plastic tubing are also useless because after the tubing has been straightened in  
the bronchoscope during insertion it takes time for it to return to its original curve.  
The tubes shown here return to their normal curve immediately after passing the  
distal end of the bronchoscope.

The spring and rubber tubing (B) is used for the right middle lobe bronchus (Figs  
62 and 67) and the basal bronchus of the lower lobe (Fig. 53), the simple curved  
tubing (C) is used for the right middle lobe or lingular bronchus (Fig. 59) and  
the curved tubing with a hook (D) is used for the right middle lobe or lingular bronchus (Fig. 59) and

lobes directed toward the apex.

E The straight rubber tubing with its two canals. The spring is inserted into the  
canal with the dead end. (Instruments shown are made by George P. Filling & Son  
Company, Philadelphia.)

are indicated. In the medical literature five methods of aspiration are described.

1. In the so-called *one catheter method* a urethral catheter or thin rubber tube is inserted through the nose into the bronchi. This catheter or tube can be kept in the airway for days and permits aspiration many times a day. This is the simplest and gentlest method of bronchial aspiration but it has two disadvantages: (a) The patient very easily expels the tube and (b) the irritation of the tube may cause laryngospasms and severe dyspnea.

2. In aspiration with *tracheal intubation* a Magill endotracheal tube of the proper size is introduced into the trachea and its distal end withdrawn through the nostril. This tube can be kept in place for several days without danger of laryngospasm and suffocation. Through this tube a thinner catheter or tube can be inserted into the bronchi for aspiration. The endotracheal tube gives adequate space for respiration; it can be kept open by lavage with saline solution and if necessary oxygen and carbon dioxide can be administered.

This would be a good method for keeping the airways clear but it too has some disadvantages: (a) The withdrawing of the relatively wide Magill tube through the nose may cause severe nasal bleeding; (b) the cough because of the tube irritation in the larynx and trachea may cause elevation of the blood pressure and recurrent nasal bleeding; (c) the prolonged intubation may cause laryngeal edema (especially in children), mucosal necrosis and granulation and (d) mechanical irritation of the lower airways may give rise to a vagus reflex and cause narrowing of the coronary arteries.

3. A satisfactory and safe method is *laryngoscopic aspiration* whereby a flexible but semirigid (of harder rubber or silk woven) aspirating tube is inserted through the direct laryngoscope and larynx into the bronchi. If the cough reflex is present the tube need not be inserted deeper than the level of the main bronchus because the laryngeal squeeze brings secretion up to this level in synergy with the mechanical aspiration. Chevalier Jackson and C. L. Jackson<sup>4</sup> call this method "synergetic direct laryngoscopic aspiration." Sometimes this method is inconvenient however for reasons given under method 4 below.

4. The usual routine method is *bronchoscopic aspiration*. This method is very often used if tenacious secretions, solidified masses, fibrinous exudates, crusts, etc. must be removed by visual guidance or if bronchoscopic examination is needed for diagnostic reasons.

Laryngoscopic and bronchoscopic aspirations are satisfactory methods

copy may cause too much discomfort to the patient after thoracic trauma and abdominal or thoracic surgery, or in other severe morbid conditions

5 When laryngoscopic or bronchoscopic aspiration are not advisable, *tracheotomy* is indicated. Through the tracheal cannula aspiration may be performed as many times as necessary, at times by nurses, and all the difficulties of the repeated aspiration may be avoided. With tracheotomy the dead space of the nasal and pharyngeal cavities is eliminated; this is a great advantage in many severe morbid conditions, such as tracheobronchial secretory obstructions in association with tetanus, brain injury, cerebrovascular accidents, neurosurgical procedures, drug poisoning, eclampsia and botulism and in postoperative conditions and debilitated patients.

In chronic respiratory disorders (advanced emphysema, excessively suppurative diseases of the lungs, pancreatic fibrosis, hyaline membrane syndrome, bulbar poliomyelitis, etc.) a permanent tracheocutaneous fistulization, by the so-called "tracheal fenestration" method<sup>21</sup> may be performed. The fistula is guarded by skin valves which provide a means of ready access to the bronchial tree when open and reestablish the normal tracheal air tract when closed. The valves may be opened repeatedly for aspiration of secretion and for a longer period of time to ease respiration.

These five methods are used to reestablish the defensive powers of the lungs. After a relatively short period, the normal functioning of peroral drainage by ciliary action and effective cough returns, and the patient feels much better. However, it would be a great error to rely on aspiration alone. Other medical care and management must be administered, such as fresh air, light diet, vitamins, antibiotics, and other systemic medications.

### *Endoscopic Treatment of Postoperative Bronchial Fistulas*

These fistulas are among the severe postoperative complications of pulmonary resections and, in a certain percentage of conditions, appear even when the most careful operative technique has been used. A fistula may close by itself, but to hasten the process bronchoscopic applications of silver nitrate, phenol, potassium hydroxide, trichloroacetic acid, electrocauterization, or diathermy are recommended. By these methods good results have been obtained in bronchial dehiscence of less than 3 mm and abscess behind the fistula less than 2 cm in diameter. In experiments with dogs Roggen, Derivaux and Duprez<sup>22</sup> obliterated bronchi from 7 to 8 mm in diameter with a 20 per cent potassium hydroxide solution. They believe this method applicable also in man.

In a consideration of treatment, the bronchial fistulas may be classified in three groups, as follows:

- 1 The abscess cavity is small, about 1 or 2 cm in diameter, and the

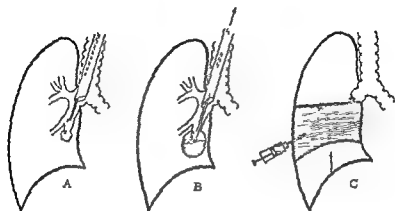


FIG 34 Bronchoscopic treatment of postoperative bronchial fistula A When the cavity is about 1 cm in diameter B When the cavity is 3 to 4 cm in diameter C When empyema is present

suppuration is retained by the suture in the opened bronchial wall rather than by the pathologic process in the cavity. After removal of the suture and by one or two administrations of caustic the dehiscence generally closes (Fig 34A).

2. The abscess cavity is 3 to 4 cm in diameter, is located centrally in the hilus and discharges excess secretions. This is the most common appearance of a bronchial fistula because the empyemic cavity shrinks in most cases to the hilus creating a fairly large abscess. The external approach to this cavity by thoracentesis is very difficult and may be dangerous in an area surrounded by major blood vessels. External drainage of this type of abscess is even more complicated.

The basic factor in the cure of these abscesses is the elimination of the suppuration. This elimination may be completed by endoscopic treatment. With bronchoscopy, sutures and granulation tissue are removed from the bronchial fistula, a urethral catheter is inserted into the cavity, and the secretions are aspirated and irrigated with antibiotic solutions (25,000 units of penicillin and 100 mg of streptomycin in a 5 ml saline solution) through the catheter. The bronchial wall in the fistula is touched with any of the previously mentioned caustics (Fig 34B).

It must be emphasized that in these conditions it is most important to stop the infection and suppuration in the abscess cavity. Closure of the bronchial dehiscence with caustics is of only secondary importance. By this simple procedure many of the bronchial fistulas are cured.

3. This group is represented by empyemas extending to the chest wall and communicating with the airways through the fistula. In these conditions, the empyema is eliminated by thoracentesis.



and external drainage, and the bronchial dehiscence is treated with caustics (Fig 34C)

The surgical closure of the bronchial fistulas is discussed in books and publications on thoracic surgery and is not the subject of this discussion

## INDICATIONS AND CONTRAINDICATIONS

### *Indications*

The field of chest diseases in which bronchoscopy is indicated either for diagnostic or therapeutic reasons, is continually expanding. Today nearly all the unexplained chest diseases may require bronchoscopy. The bronchologist reveals conditions of the bronchial mucosa, the bronchial wall, and the pathologic action of the bronchi. With biopsy, aspiration of secretion, and segmental lavage he obtains material for histologic, cytologic, and bacteriologic examinations. He removes obstructing material—thick secretions, foreign bodies, ruptured granulation tissue, cheesy debris, broncholiths, and benign tumors—and he performs aimed bronchography. The bronchologist assists the chest surgeon prior to operation, during operation, and after operation by cleaning the airways. He helps the pneumologist, the roentgenologist, and other physicians to localize diseases. Therefore, it is nearly impossible to lay down exact rules as to every specific situation that calls for bronchoscopy. Actually, bronchoscopy is indicated in every thoracic disease in which the diagnosis is uncertain and in which the medical treatment is inadequate.

The *absolute indications for bronchoscopy* are stenosis or obstruction of the trachea or bronchi, purulent sputum, hemoptysis, and presence of a foreign body. In these cases omission of bronchoscopy involves severe consequences. Further details will be given in the discussions of the various diseases, in the chapters that follow.

### *Contraindications*

If bronchoscopy is really needed, contraindications do not exist. It is well known that a foreign body must be removed from the lung even in a patient with advanced cardiac disease. Bronchial obstruction must be removed, if the patient is debilitated, by pulmonary suppuration, and, more important, bronchoscopic aspiration or lavage (with antibiotics) may help a patient through a critical situation, e.g., a pulmonary abscess. Bronchoscopy is imperative in cases of acute laryngotracheobronchitis or bronchial asthma when thick, tenacious secretions occlude the bronchi.

However, bronchoscopy is postponed in severe cardiac conditions or hypertension, in any acute inflammation in the respiratory tract, and in severe hemoptysis (Chap 10). Most writers oppose bronchoscopy in

cases of aneurysm because of the danger of rupture. The author believes that for various diagnostic reasons bronchoscopy must often be performed in these cases. Naturally in the presence of an aneurysm the bronchoscope is inserted with the greatest care and biopsy is inadvisable.

A word is appropriate regarding the cooperation between the physician who requests bronchoscopy and the bronchologist. When a physician requests a bronchoscopic study, he has already performed at least a general examination and obtained one chest x-ray. Without these he may not even think of bronchoscopy, except in an emergency. Bronchoscopy may never be his first thought in the course of his examination. The bronchologist must know the referring physician's problem. The referring physician has a right to expect quick and proper work, a precise report and help from the bronchologist, if he supplies the bronchologist with all the data on past medical history and previous examinations which sometimes are available only from a family doctor.

### Summary

The instruments used by the bronchologist are the bronchoscope, magnifying instruments, auxiliary instruments, photoapparatus and operating room equipment.

Prior to bronchoscopy the patient's physical condition and chest organs must be thoroughly examined; organic diseases must be excluded.

Where local anesthesia is used, premedication must be given at least 1 hour prior to examination. The drugs generally used are Nembutal and Demerol as sedatives and morphine and scopolamine as opiates. Atropine also may be administered for diminution of the bronchial secretion. The local anesthetics are cocaine (1, 4 or 10 per cent), Pontocaine (0.5 or 2 per cent) and Nylodine (1 or 2 per cent).

For general anesthesia in children, premedication and the open ("open drop") method of administering ether are generally used. In adults, curare or succinylcholine as relaxants and Pentothal Sodium given intravenously as general anesthetic are used, and these may be followed by nitrous oxide and oxygen or other agents.

The generally accepted practice in bronchoscopy is to place the patient in the dorsal recumbent position. The bronchologist first exposes the glottis by introducing the tube into the patient's mouth along the right side of the tongue. The trachea is the second subject of the bronchoscopic examination, after which step by step every bronchus must be inspected. Bronchoscopy must always be exact but as short as possible. Bronchoscopic views are shown in Figure 32.

The delicate, vulnerable tissues of an infant or young child may be confused during bronchoscopy. Therefore the examiner endeavors to avoid repeated introduction of the tube and cuts the duration of bron-

choscopy as short as possible without sacrificing the precision of the examination

The most sensitive region in infants and young children is the subglottal area of the larynx which can easily be injured by an oversized tube. From this subglottal edema may arise with its alarming symptoms. The largest tube which readily passes through the larynx and yet yields the widest view must be selected.

The inexperienced person should not perform bronchoscopy in infants and young children without skilled assistance. However in the hands of an experienced specialist bronchoscopy is an easy and safe procedure for infants and even for the newborn.

Simple bronchography has been a well known and frequently used procedure for several decades in clinical routine. The following points are worth remembering:

1. Bronchography requires deeper and more accurate anesthesia than bronchoscopy.

2. Filling of the bronchial tree should be controlled by fluoroscopy and exposure should be taken at optimal filling in every case.

3. Filling of bronchi in a lobe or segment may be inadequate or even absent for several reasons. In these instances aimed bronchography is indicated. Aimed bronchography may be utilized in every instance of filling defect (dead tree effect) involving a pulmonary unit with positive or even negative x-ray findings. Surprising results are often obtained.

In every case of continually accumulated secretion in the lower airways repeated aspirations of the secretions are indicated. The methods used are the one catheter method, aspiration with tracheal intubation, laryngoscopic aspiration, bronchoscopic aspiration and tracheotomy or tracheal fenestration. The defensive power of the lungs can be reestablished by these procedures.

Bronchial fistulas are among the severe postoperative complications of pulmonary resections. In case of a small cavity due to a fistula removal of the sutures and application of caustic may be adequate. If the abscess cavity is 3 to 4 cm in diameter bronchoscopic aspiration and irrigation of the cavity itself should be performed. Many fistulas have been cured by this method. If empyema is present external drainage or surgery is indicated.

There are no strict rules regarding the circumstances under which bronchoscopy is appropriate. The field of chest diseases in which bronchoscopy is indicated is expanding continually. Actually bronchoscopy is indicated in any thoracic disease when the diagnosis is uncertain and when the treatment has been inadequate. Contraindications do not exist if bronchoscopy is really needed. However endoscopic examination should

## Diseases with Valvular Respiratory Mechanisms

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A *valve* is generally a structure which regulates the flow of a gas or liquid. Some valves permit flow in both directions in living organisms; these are the sphincteric closing mechanisms. The sphincter of the cardia permits flow toward the stomach in ingestion and outward in vomiting or eructation. Most valves permit material to pass in only one direction in living organisms; these are the valves of the heart and the vessels. In connection with respiration, valves exist chiefly in pathologic conditions. However, the glottis also acts as a particular valve when in coughing it closes and then suddenly opens; this action will be referred to later in this chapter under Trauma of the Lung Tree.

Valves connected with respiration are formed in valvular bronchial stenosis, inflamed cysts after tracheotomy in trauma of the lung tree, and in some other diseases. Diseases with valvular mechanisms caused by injuries to the chest wall are outside the scope of this discussion.

Valvular bronchial stenosis was discussed in detail in Chapter 3 along with other respiratory mechanisms.

### INFLATED VALVULAR CYSTS

All inflated cysts have in common the fact that they are inflated by a valvular respiratory mechanism, but the genesis of this mechanism and that of the cyst itself is variable. It may be parasitic (echinococci) or it may be tuberculous; it may originate from an abscess, carcinoma, or an inflammatory lesion of small bronchi or bronchioles, or from rupture of the pulmonary parenchyma. Thus cysts with varied origins and formations, such as pleural bullae, emphysematous bullae, or pneumatoceles, may be inflated and become large or even of giant size. Questions relating to inflated tuberculous or nontuberculous cavities, circumscribed emphysema, inflated pneumatocele caused by bronchial carcinoma, or cystic lung are discussed in the chapters dealing with those diseases.

Other cysts originating from the pulmonary tissues can be classified as bronchial or alveolar

### **Bronchial Cysts**

The bronchial cysts originate from accessory bronchial buds. The inner surface of the cysts is covered with a mucosa similar to that of the bronchi. They are generally pedicellated; the pedicle is attached to a larger bronchus somewhere in an interlobar fissure. These cysts are solitary and rare, more often unilateral than bilateral.

The following example shows the diagnostic procedure in a case of a bronchogenic cyst.

**CASE 3** A female infant became dyspneic at ten months of age. On admission to the hospital 2 months later protrusion of superior intercostal space of the chest and circular indrawing at the diaphragmatic level were observed. The child was severely dyspneic.

In the x ray (Fig 35A and B) the heart was displaced to the right side; on the left side the lung markings were absent; only a small triangular density appeared in the cardiophrenic angle; on the right side a curved, laterally convex, ribbonlike density extended from the third to the tenth ribs.

On thoracentesis of the left chest cavity about 150 to 180 ml of air escaped under pressure, and in consequence respiration improved. On the following day another puncture had the same result. Bronchoscopic findings, except for observation of displacement of the bronchial tree, were negative.

Valvular pneumothorax was suspected, and bronchograms were taken. Bronchograms (Fig 35C and D) showed that the entire left lung was compressed upon the left diaphragm; the upper lobe located anteriorly and the lower lobe posteriorly. From the elongated left main bronchus, before its division, a channel directed forward and laterally led into the cyst cavity (see arrows in Fig 35C and D).

The diagnosis was now established. A valvular bronchogenic giant cyst communicating with the left main bronchus was located in the upper and middle portion of the left chest cavity with displacement of the mediastinum and heart extending also into the upper anterior portion of the right hemithorax.

Chest surgery was recommended, but because the patient was very undernourished it was postponed.

Almost every bronchial cyst indicates surgical resection.

### **Alveolar Cysts**

The walls of alveolar cysts are created by compressed alveoli without epithelial lining. Actually these are pseudocysts. This type of cyst is always aerated, while a bronchial cyst is not always aerated.

Characteristic symptoms are absent in uncomplicated cases of pulmonary cysts. Sex and age are not characteristic factors. Almost always acci-



FIG. 35 Anteroposterior and lateral x rays of a one year old girl (see Case 3) A On

arrows)

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FIG. 36 X-ray films of a two-year-old girl (see Case 4) show an inflated giant cyst in the right lung. A The heart and mediastinum are displaced to the left; no pulmonary markings are noted in the right chest cavity. B Bronchograms showing the right bronchial tree compressed against the mediastinum and stretched not collapsed to the hilus. C is usual in cases of pneumothorax; inflated giant cyst was the diagnosis. D Anteroposterior x-ray film shows the former cyst divided into several smaller loculi. E F Lateral and anteroposterior x-ray films showing that the giant cyst has decreased; it appears as a smaller oval thick-walled cavity. G The formerly compressed parenchyma has expanded, and the cyst has decreased into a still smaller cavity. H Anteroposterior x-rays showing the absence of the cyst.

dental circumstances (complications, serial examinations, etc.) call attention to them. Complications may be bleeding, cough, or dyspnea. Valvular action often inflates these cysts, causing respiratory or circulatory disturbances. The wall of the inflated cyst may also rupture, resulting in valvular pneumothorax with or without interstitial emphysema. Valvular cysts are often confused with valvular pneumothorax. The majority of inflated valvular cysts are alveolar. They arise frequently after pulmonary inflammation, when mucosal congestion or thick secretion causes valvular action. They are very interesting examples of a combination of primary infection and the consequent mechanical function, which is a basic principle in pulmonary pathology. In inflated cysts the inflammation causes the stenosis, and the mechanical effect causes the inflation.

The interior of a giant alveolar cyst may be empty or filled with a web of inflated alveoli unrecognizable by x-ray. The web becomes visible if the wall of one part of the alveolar structure ruptures; the other alveoli collapse and create thicker layers which project definite shadows on the x-ray film (Figs. 36D and 38C).

The confusion between valvular pneumothorax and giant cyst with alteration of the cyst's interior is demonstrated in the following case.

**CASE 4** A two-year-old girl suffered in March, April, and May from pertussis, measles, chickenpox, and bronchopneumonia. Closely following the last disease, complete pneumothorax and atelectasis of the upper lobe was diagnosed on the right side. The disease was considered of tuberculous origin, and streptomycin was administered.





A



C



D



E



G



H



I

(See caption on facing page)

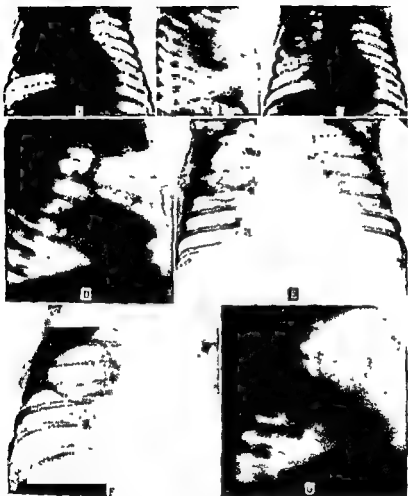


FIG 37 X ray films of a premature infant (see Case 5) A Atelectasis of the right upper lobe with pneumonia (increased in volume) B C Most of the atelectatic lobe has cleared only anterior segment remaining airless but in its center a pea sized

On admission to the hospital in June the child was debilitated and thin. The heart and mediastinum were displaced to the left side, and no pulmonary markings were noted in the right chest cavity. Reaction to intracutaneous tuberculin test and results of bacteriologic examinations of the sputum for acid fast bacilli were negative.

X rays taken in May were unaltered for the succeeding 4 weeks (Fig 36A). On June 25, bronchoscopy was performed and bronchograms taken. Bronchograms showed the right bronchial tree pressed to the mediastinum and stretched, not collapsed to the hilus as is the usual case in pneumothorax (Fig 36B and C), therefore, inflated giant cyst was the diagnosis. On July 11 the large cavity, which appeared empty, was shown to be divided into several smaller cavities, confirming the diagnosis of giant cyst instead of valvular pneumothorax (Fig 36D). On August 6, the cyst was shown to have decreased and become a smaller, oval, thick walled cavity (Fig 36E and F). By October 13, the compressed parenchyma of the right lung expanded, the cyst decreased into a small cavity (Fig 36G), and by November 18 it had disappeared entirely (Fig 36H).

After bronchopneumonia a valvular inflated cavity occupied the entire right chest cavity. This giant cyst was considered at first to be a valvular pneumothorax. Bronchograms, and the subsequent bizarre variations of smaller cysts which developed inside the giant cavity, clarified the diagnosis. The valvular cyst disappeared within 5 months.

The successive events of infection, inflammation, and valvular mechanism are obvious in this case. The relation of these lesions will be more evident in the following case.

**CASE 5** A premature infant was transferred to a hospital for premature infants in Budapest. He weighed 33 oz in his second week. The entire right upper lobe became atelectatic and inflamed (Fig 37A). By the fourth week, most of the atelectatic lobe had cleared, only the anterior segment remained useless, but in its center a pea sized aerated cavity appeared (Fig 37B and C). Within a few days the atelectasis completely cleared, but the cyst slowly increased (Fig 37D and E) and during the next 3 months inflated to the size of a small walnut (Fig 37F and G). This lesion did not retard the infant in his development. He was discharged from the hospital when he was five months old at a weight of 112 oz, with the cyst persisting. The next examination, 2 months later, showed absence of any lung pathology.

In the above two cases the inflated alveolar cysts disappeared spontaneously after the inflammatory stenosis ceased to exist. This is the usual history of an inflated alveolar cyst. However, in some cases the cysts resist conservative treatment and persist until permanent external suction (by thoracentesis) or surgery is performed. For example

**CASE 6** A three-year old boy had been dyspneic for 2 years. There was a questionable diagnosis of valvular pneumothorax or cyst (Fig 38A and B).



FIG 39 X rays of a four year-old boy showing mediastinal emphysema and interstitial emphysema around the bony chest wall (see arrows) after tracheotomy at too low a level. A Lateral B Anteroposterior

Therefore air finds its way only through the tracheal stoma however with the skin wound closed around the cannula its exit is obstructed and the air is forced into the tissues of the neck. This valvular mechanism is well known to every surgeon.

2 In certain cases cervical and mediastinal emphysema arises even if the wound is dressed regularly with an open pick. Emphysema occurs chiefly in childhood if the loose mediastinal connective tissues are opened during the operation. In these cases during inspiration the patient aspirates air into the mediastinum through the tracheal stoma and skin wound. In forced expiration and particularly during cough the extruding tissues obstruct the opening between the trachea and sternum thus the air not only becomes trapped but also is forced further into the loose mediastinal tissues (Fig 39). The cough is stimulated by the emphysema the cannula and also by the stretching of the mediastinum therefore in many cases the cough is continual. The cough increases the aspiration of air into the mediastinum with deepening of inspiration and spreads the emphysema by increasing the expiratory pressure. Air fills the mediastinum extends to the neck head chest and then the whole body.

To impede the progress of the emphysema a tight tampon should be inserted into the jugular opening of the mediastinum and the cough should be decreased with sedatives in children or codein in adults in very severe cases mediastinal drainage may be necessary.

3 When in cases of laryngeal or tracheal stenosis the patient exhales fully or coughs hard the stretched pulmonary parenchyma may rupture causing interstitial emphysema and its complications (pneumo-



FIG 38 X-ray films of a three-year-old boy (see Case 6) A B Inflated valvular cyst in the right chest cavity herniated into the left chest cavity C After permanent external suction several smaller loculi arose inside the giant cyst but the cyst itself did not decrease D The surgically removed cyst

Examination started with bronchoscopy and bronchography the results of which suggested valvular cyst The lesion did not respond to bronchoscopic aspiration and lavage therefore permanent external suction was instituted Because of this suction several smaller cysts arose inside the giant cyst but the cyst itself did not decrease (Fig 38C) With external surgery the giant cyst (Fig 38D) which arose from the parenchyma of the anterior segment was removed The major bronchi were absent in the adjacent parenchyma of the remaining lobe as well as in the surgical specimen

### VALVULAR MECHANISMS AFTER TRACHEOTOMY

Tracheotomy may cause three types of valvular mechanisms

- 1 Sometimes subcutaneous and mediastinal emphysema may arise if the wound is stitched tightly to the cannula During expiration and more noticeably in coughing the air cannot be expelled through the stenosed larynx upper trachea closed glottis or perhaps the obstructed cannula

juries, simultaneous and isolated, but mainly in the isolated ones. The chief causes of the isolated traumas are as follows:

1. After deep inspiration, when lungs are filled to capacity, a person, e.g., an athlete, while exercising, may spasmodically close his glottis. In this state the "lung tree" is expanded by the trapped air. If at that point a strong external pressure is exerted on the thoracic wall, the expanded trachea or bronchi may rupture.

2. The blast from an explosion near the open mouth may penetrate into the lung and rupture the trachea or one of the major bronchi.

3. A fall from a high place to a hard surface (e.g., from a window, scaffold, automobile, or airplane) may result in avulsion of a lung from the hilus by its own weight and rupture of the trachea, esophagus, or main bronchi.

4. Crushing injuries, such as those incurred by being run over, by a cave in, by collision or by compression between buffers, may compress the chest in such a way that the chest wall itself does not break but inner injuries arise. This type of trauma occurs particularly in childhood when the thoracic cage is relatively soft and flexible.

5. The tracheal or bronchial wall may be perforated by incorrectly or ineptly performed bronchoscopy (Fig. 41).

The rupture in most cases is transverse. Longitudinal or oblique ruptures rarely occur because the transverse cartilages are resistant.

**Signs and Symptoms.** The clinical appearance of isolated tracheal and bronchial rupture is unique, well differentiated and dramatic. The signs and symptoms are as follows:

**Shock**, generally appearing in cases of external injury, is not always observed in cases of isolated tracheobronchial rupture. Severe shock is generally caused by trauma of the thorax; death occurs as the result of shock and rarely from dyspnea or suffocation. A detailed discussion of shock does not fall within the boundaries of this subject.

**Pain in the chest** is always present.

**Impairment of the voice** is often due to injury of one of the recurrent nerves.

**Sputum** usually is foamy and sanguinous.

**Dyspnea** is the cardinal symptom resulting from the stenosis.

**Interstitial emphysema** is always present in rupture of the trachea or of a major bronchus and spreads very rapidly and characteristically. It expands within minutes to the neck, chest, face, and scalp and perhaps also into the pleural, pericardial or abdominal cavities, and to the whole body. The quick spread alone may cause death within 10 minutes. Spread of emphysema is particularly rapid in childhood; it occurs from scalp to the toes, air blebs have been observed even under the nails; they penetrate into the joints and muscles, and the musculature may be elevated from

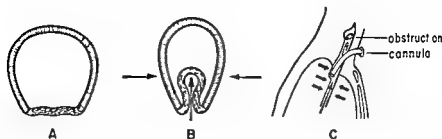


FIG 40 The horizontal section of a trachea A During normal respiration B During cough the C shaped cartilages bend inward and the soft paries membranaceus invaginates into the tracheal lumen (From Stut<sup>100</sup>) C During cough the tracheal invagination obstructs the distal lumen of the cannula and because of the laryngeal obstruction the trapped air may rupture the pulmonary tissues

thorax pneumopericardium pneumoperitoneum) The reason for this is obvious when airways are obstructed but it is not easily understood if it occurs when the dyspnea is relieved by tracheotomy and the lumen of the cannula is free

It is well known that during forced expiration and cough the C shaped cartilages of the thoracic trachea bend inward and the soft paries membranaceus tracheae invaginates into the tracheal lumen (Fig 40A and B) This phenomenon is called *tracheal invagination*<sup>100</sup> The bronchologist frequently observes it in infants and children during bronchoscopy After tracheotomy the child frequently coughs hard and continually In inspiration the lumen of the cannula is free but in expiration and especially during cough the invaginating paries membranaceus tracheae may cover the inner orifice of the cannula and obstruct it by valvular action (Fig 40C) The trapped air may rupture the stretched parenchyma Probably this is the explanation of this otherwise poorly understood process Further details are given below under Trauma of the "Lung Tree"

## TRAUMA OF THE "LUNG TREE"

Injury to the cervical trachea is not rare its diagnosis and treatment are however in the field of neck surgery and are therefore not discussed here

The thoracic trachea is hidden behind the strongest portion of the bony chest cage The bronchi are protected by this bony wall and in addition by thick layers of soft tissue therefore the thoracic trachea and bronchi become traumatized not so often by direct injury to these organs alone but more often in association with injury to the thoracic wall and other organs within the thorax However isolated trauma of the bronchi also may occur The apparent rarity of this injury is due at least partly to lack of statistics<sup>4</sup> The bronchologist is interested in both forms of in

juries, simultaneous and isolated, but mainly in the isolated ones. The chief causes of the isolated traumas are as follows

1 After deep inspiration, when lungs are filled to capacity, a person, e.g., an athlete, while exercising, may spasmodically close his glottis. In this state the "lung tree" is expanded by the trapped air. If at that point a strong external pressure is exerted on the thoracic wall, the expanded trachea or bronchi may rupture.

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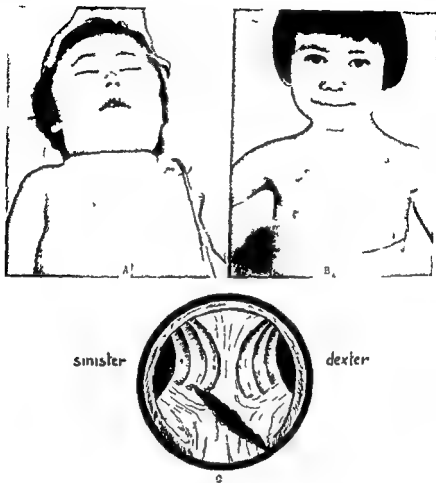


FIG. 41 A four year old girl A The child inflated by interstitial emphysema after bronchoscopic perforation of the trachea above the carina tracheae left valvular pneumothorax is under external suction B Eight days after rupture was sutured by external surgery C Bronchoscopic view of the position of the perforation

the bones. This is called *general interstitial traumatic emphysema* (Fig. 41). It arises in the following way. In inspiration the wound opens and air is inspired into the mediastinum. In expiration the wound collapses and air becomes trapped. The pronounced spread of the emphysema is partially explained by the excited cough which increases the inspiratory suction and the expiratory pressure, but the cough produces also another phenomenon. During a cough the glottis becomes closed and the "lung tree" is confined under strong pressure. The wound opened by this pressure permits air to enter the mediastinum. This is also a valvular action.

ie the particular *valvular action of the glottis*. Thus there is a *double valve*. One valve is represented by the wound in the "lung tree" itself and the other by the glottis. During normal respiration mediastinal emphysema occurs only in inspiration but during inspiration of air to the chest, the double

air to the chest, the double

Empyema must to the neck then over the chest (Fig. 41). Over the chest fine synchronous crackling with the pulse is audible. Heart dullness and the apex impulse are diminished or absent and dull heart sounds are noted. Alternating and diminished breath sounds are perceived. Equivocal descent of the liver dullness respiratory and swallowing impairment pneumothorax and pneumo pericardium also may arise.

The respiration is wheezing, twanging sometimes whistling. Expiration is impulsive and interrupted.

Emphysema may extend also to the pharynx, mouth and even into the nasal pharynx closing the choanal openings. The voice therefore of the injured patient is a strange deep closed nasal voice. On battlefields trauma of the airways and mediastinal emphysema were diagnosed some times only from this strange voice.

X-ray films show the mediastinum wide and containing large air blebs. If emphysema has already extended subcutaneously a transparent layer of air can be observed between the bony chest wall and the skin of the chest (Fig. 39).

The diagnosis from these signs and symptoms is generally easy.

In tracheobronchial perforation or where it is suspected a bronchoscopic examination should be made at once if the patient's condition permits. By bronchoscopy accurate diagnosis and localization may be stated for the thoracic surgeon who will close the perforation without delay. Stenosing changes—dislocation protrusion of the ruptured edges—may be not only inspected but also possibly corrected to decrease dyspnea and retard development of the emphysema. Blood and secretion may be aspirated. Atelectasis frequently is due to retained secretion but may be of reflex origin and the genesis can be determined only by bronchoscopy.

In cases of chest injury the duty of a bronchologist is to restore and maintain free respiration equally before during and after operation. For some days after the trauma deep breathing and expectoration are obstructed the accumulated secretions must be eliminated. Particular attention should be paid to the cleanness of the airways if the patient undergoes surgery.

Prognosis. The prognosis is good in cases of minor trauma without mural perforation. The penetrating injury urgently calls for external

rupture is circular mediastinal and pulmonary tissues protrude into the ruptured bronchial ends increasing the stenosis and possibility of infection pneumonia pleuropneumonia empyema or abscess may arise in the pulmonary parenchyma distal to the stenosis In these cases the prognosis is poor

## OTHER DISEASES

Valvular action also takes part in the origin of emphysema The valvular action inflates the alveoli and hinders the respiration especially the expiration because of stenosis of the minor bronchi This stenosis is caused by inflammatory or allergic congestion of the mucosa This phenomenon is seen in cases of *emphysema due to chronic bronchitis* and *bronchial asthma* By this mechanism circumscribed emphysemas and delicate bronchiectasis may arise Some writers talk directly about "bronchial emphysema" <sup>1 20</sup>

The part played by valvular action in the development of bronchiectasis is discussed in Chapter 9

### Summary

Valvular mechanisms connected with respiration arise in valvular bronchial stenosis inflated cysts after tracheotomy in trauma of the lung tree and in some other diseases

Inflated cysts originating from pulmonary tissues may be bronchial or alveolar

Bronchial cysts originate from accessory bronchial buds They are true cysts and have their own wall which is lined with bronchial mucosa

Alveolar cysts are pseudocysts their walls are composed of alveoli which are compressed by the inflated pulmonary tissues They occur most frequently after pulmonary inflammation and are the most interesting examples of the combination of primary infection and consequent mechanical function The valvular bronchial stenosis is caused by the inflammation the inflation by the valvular mechanism

Almost every bronchial cyst indicates surgical resection most of the alveolar cysts disappear spontaneously

Tracheotomy may cause three types of valvular mechanisms (1) The expired air is forced into the tissues of the neck and mediastinum when the skin is tightly closed around the cannula (2) Mediastinal emphysema may occur even if the wound is dressed with an open pack regularly in this case the wound penetrates into the loose mediastinal tissues and the forced inspiration aspirates air into them (3) So called "tracheal invaginations"

tion " acting as a valve obstructs the distal lumen of the cannula. The trapped air during forced expiration or cough may rupture the pulmonary parenchyma and through pulmonary interstitial emphysema cause pneumothorax pneumoperitoneum or pneumopericardium. Its clinical appearance is unique well differentiated and dramatic resulting in general interstitial traumatic emphysema. Valvular action also takes part in development of pulmonary emphysema due to chronic bronchitis or bronchial asthma. It may play a role in the origin of bronchiectasis.



# Infectious and Allergic Bronchitis

· 6 ·

## ACUTE INFECTIVE LARYNGOTRACHEOBRONCHITIS

Laryngotracheobronchitis today is considered by more and more writers<sup>12</sup> as a special pathologic condition in spite of the fact that secretions contain not just one type of pathologic bacteria but mixed organisms. It occurs chiefly in infants and children and is frequently complicated by pneumonia or bronchopneumonia.

Although the disease is fairly rare it must be thoroughly understood because of its severity. Its relative frequency in childhood and its characteristic symptoms can be explained by the absence of immunity the looseness of tissues the narrow lumens of the airways weak cough reflexes in infants and young children and the characteristically thick secretion. No other disease may be observed in the advanced state. These crustlike secretions are characteristic of the disease and are its chief danger. This condition is very serious in an infant and complications occur because the infant with his weak cough reflex and ineffective expectoration is unable to bring up the thick secretion from the narrow airways.

The chief symptoms are the debilitation of the patient and suffocation. Diagnosis is made or confirmed by direct or (in adults) mirror laryngoscopy. The laryngeal mucosa is dark red. In the beginning the secretion is serous later mucoid or purulent and finally crustlike and scattered in sticky spots.

Therapy will be discussed only from the bronchologist's point of view. If the pediatric treatment, i.e. the fight against infection and dyspnea brings inadequate results then there is no time to wait. The infant must be relieved of the thick secretion and air permitted to enter the alveoli. First gentle but effective aspiration of the secretion must be performed. If this procedure is required frequently tracheotomy is indicated and the secretion eliminated through the tracheal cannula. If this treatment is also insufficient then the thick tenacious or crustlike secretions must be removed or eliminated (perhaps with forceps) from the bronchial orifices.

bronchoscopically. Instillation of 1 per cent sulfoacid sodium solution (in an 0.5 to 1 per cent wetting agent) or inhalation with Alevaire softens the crusts and makes them easy to remove. Unless evacuation of the airways is accomplished, the infant suffocates, or if he survives the severe disease pulmonary complications (pneumonitis, abscess), permanent cicatricial bronchial stenosis, or bronchiectasis may develop.

## CHRONIC AND DEFORMING BRONCHITIS

The etiologic factors in chronic and deforming bronchitis are repeated acute inflammations of the bronchi, chronic purulent inflammations of the upper respiratory organs (nose, paranasal sinuses, nasopharynx, tonsils), circulatory disturbances, allergy, and prolonged irritations. Irritations may be the result of smoke or dust inhalation, aspiration of chemicals or excessive smoking or consumption of alcohol.

Of the group of irritating materials, dust inhalation is the most common. The dust may penetrate into the alveoli and further into the pulmonary parenchyma. The protective mechanism of the respiratory organs tries to resist this process. Normally about 50 per cent of the dust is retained by the nasal mucosa, 25 per cent is kept by the mucous membrane of the lower respiratory tract, and from 3 to 15 per cent returns to the outer world. Thus only from 10 to 15 per cent of the dust enters the alveoli if the dust granules are less than  $4\ \mu$  in diameter (this being the diameter of the respiratory bronchioles). The penetration of the dust is furthered by nasal respiratory disturbances (septum deviation, nasal polyp, etc.) and by an abnormally wide nasal cavity (ozena). The dust has a mechanical (foreign body) effect, it carries infection and may irritate chemically, as well.

The bronchial glands react to these irritations by increasing their secretions which serve as a breeding ground for the bacteria. This is the start of inflammation. If the bronchitis is prolonged, anatomic and functional changes occur. As a result of inflammation the epithelium loses its cilia, the single layered epithelium becomes multilayered, and the cleansing is retarded.

**Diagnosis.** Not long ago it was held that nearly all patients with persistent coughs had chronic bronchitis. If the chest x-ray was negative and pulmonary tuberculosis could be more or less excluded, physicians diagnosed the case as chronic bronchitis.

The broad application of bronchoscopy proved that such an approach was no longer adequate. The bronchologist diagnosed bronchostenosis in 43 per cent, bronchiectasis in 23 per cent, pulmonary tuberculosis in 8 per cent, and lung tumors in 5 per cent of the cases of so called "chronic

bronchitis." Chronic bronchitis was found bronchoscopically in only 21 per cent.<sup>42</sup>

The first duty of the bronchologist is to reveal the reasons behind the symptoms, such as a persistent cough and discharge of secretion. Next, his function is the elimination of the cause and the treatment of the inflammation. In cases of chronic bronchitis the bronchial mucosa is red, moderately swollen, velvety, and in places covered with secretions. The secretion is usually thick, transparent, and yellowish or greenish. The bronchologist aspirates the secretions, under sterile conditions, for bacteriologic examination.

The inflammatory swelling of mucosal tissues narrows or occludes the long efferent ducts of the bronchial glands and diverticula (Chap. 1). The retained secretion dilates the ducts and the acini. Delicate saccular canals occur which may persist after the disappearance of the inflammation and the reopening of the ducts. Bronchograms show these dilatations as thorns on the outer bronchial walls (Fig. 42).

If inflammation persists and increases, the canals become infected. Since the bronchial glands and diverticula extend beneath the cartilages in many places, deeper layers of the bronchial wall become affected. Small abscesses and small areas of necrosis appear. Their consequences are granulation and cicatrization. Smooth muscle reacts to inflammation with tonic disturbances, spasms and paralysis alternate. These actions destroy the muscle, and granulation and cicatrization replace the elastic fibers, thus, the bronchial wall, which has great resistance, weakens. These are the steps in the development of deforming bronchitis.



FIG. 42. Bronchogram showing small glandular diverticula in the wall of the right main bronchus in a case of chronic bronchitis.





FIG 43 Bronchograms in cases of deforming bronchitis A The spastic type B The paralytic type C The paralytic type with developing bronchiectasis

The evolution of deforming bronchitis may be observed on bronchograms. In the early phases the bronchograms show slight unevenness and spasmodic stenosis on the bronchial walls (Fig 43A). Reflexes to which the smooth muscles react are increased. In this stage of the inflammation most of the secretion is expectorated. Subsequently distortions and dilations of the bronchi prevail (Fig 43B). In this advanced stage the amount of secretion is increased, reflex stimulation because of damage to the bronchial wall decreases, expectoration of secretion is hindered. Many round filling defects appear on bronchograms because drops of secretion are mixed with the contrast medium. Finally pronounced cylindrical or sacular dilations can be seen in the bronchi, smooth muscle function is absent and drainage of secretion is diminished. The process has reached the stage of bronchiectasis (Fig 43C).

Deforming bronchitis develops chiefly in bronchi in which the secretion is retained. Thus the bronchitis is most pronounced in the bronchi of the lower lobes, next in the inferior lingular segment, followed by the approximately horizontal bronchi of the anterior and the superior lingular segments. The right middle lobe reacts with peculiar vulnerability to inflammation (Chap 7).

**Therapy.** Deforming bronchitis at first may be limited to a single segment and later may spread to the other bronchi. Naturally the more severe the inflammation is and the longer it persists, the faster it spreads and the more extensive the process becomes. This fact is important because with diminution or cure of the inflammation the extension may be prevented. First the patient is treated with the usual internal medications. Previous bacteriologic examination is very important because depending on the sensitivity of the bacteria, antibiotic therapy will be instituted and autovaccine therapy also may be administered. The bronchologist performs bronchial lavage to decrease the inflammation and

secretion. The commonly used antibiotics are penicillin (40,000 units in 5 ml of physiologic saline solution) and streptomycin (0.1 Gm in a similar solution). In cases of mixed infection these antibiotics may be used simultaneously. Bronchial lavage can be performed once or twice a week.

If, in addition to a pronounced bronchiectasis, deforming bronchitis is observed also in the neighboring bronchi, extirpation of the bronchiectatic portion must be performed as soon as possible. With the extirpation the extension of the process may be arrested. After the operation treatment of the deforming bronchitis must be continued, otherwise bronchiectasis will develop in the segments, as well.

### BRONCHIAL ASTHMA

In this disease acute hypoxemia occurs with the attacks of dyspnea. The respiratory disturbance is caused by spasm of smooth muscle of the delicate bronchioli, by allergic mucosal edema, and by the very thick tenacious secretion. Both inspiration and expiration are retarded, but because in expiration the lumens of the bronchioli narrow, expiratory dyspnea is more pronounced. Therefore, the alveoli become inflated. During an attack acute emphysema develops, with its characteristic symptoms. In young children frequently repeated attacks keep the relatively soft wall of the thoracic cage in an inflated state and deform it. Smooth-muscle spasm is caused by reflex and/or allergic stimulations.

The reflex stimulation originates from the vagal nerve endings of the larynx and the lower airways or from irritation of the trigeminal fibers caused by nasal diseases. Tracheal and bronchial nerve ends may be irritated by foreign body, tumor, perforation of lymph nodes, etc.

The allergic irritation is a consequence of sensitivity to foreign proteins. The most frequent allergens are pollens, dander from the skin of animals (horse, dog, cat, etc.), feathers, house dust, food, or bacterial protein. In allergic asthma Curschmann's spirals, Charcot-Leyden crystals, and eosinophilic leukocytes are found in the sputum. The blood also contains an increased number of eosinophilic leukocytes.

The bronchologist is interested in every member of the first group, reflex stimulations, and in the second group, allergic irritations, chiefly in bacterial asthma. The nose, throat, tonsils, and larynx of every patient with asthma must be examined. This examination is particularly important if the characteristic eosinophilic cells cannot be found in the blood and/or sputum. In these cases it must also be considered that the presence of status asthmaticus may be caused by diseases of the lower airways, foreign body, tumor, etc., and thus bronchoscopy must be performed.

Allergic asthma may be caused by the bacteria of a chronic bronchitis.



FIG 44 Chest films of a forty two year old woman with bronchial asthma. A B Atelectasis in the left lower lobe due to thick secretions of bronchial asthma. C D No pathologic condition seen 2 days after bronchoscopic aspiration (Case from the Lankenau Hospital Philadelphia)

In these cases a culture of pathogenic bacteria obtained from the secretion by sterile bronchoscopic aspiration may be made. Sensitivity tests may reveal the proper antibiotic to be used. Autovaccine also may be produced and administered.

Therapy of allergic asthma generally does not concern the bronchologist but if the extremely thick and tenacious secretions accumulate in the bronchi of a patient with asthma and cause atelectasis or dyspnea these secretions must be aspirated bronchoscopically (Fig 44). By this procedure death by asphyxia is frequently avoided.

### Summary

Acute infectious laryngotracheobronchitis is a special pathologic condition which in most cases is a very severe disease, especially in infants. Complications occur because the infant with his weak cough and ineffective expectoration is unable to bring up the very thick secretion from the narrow airways. Therapy, from the bronchologist's point of view, consists in the immediate removal of the thick secretion by aspiration, tracheotomy where indicated, and bronchoscopic removal of the crustlike secretions.

No matter what the etiology is (inflammation, circulatory disturbance, allergy, or irritation), bronchitis may undergo three developmental stages (acute, chronic, and deforming), and if this destructive process is not stopped, bronchiectasis will finally develop.

Not long ago it was held that nearly every patient who coughed persistently suffered from so called "chronic bronchitis." The broad application of bronchoscopy proved that such an approach was no longer adequate. Prebronchoscopic diagnosis of chronic bronchitis was confirmed bronchoscopically in only 21 per cent of cases.

The duty of a bronchologist is to reveal the reasons behind the symptoms, to eliminate the cause, to treat the bronchial disorder, and to prevent the development of the bronchiectasis.

Bronchial asthma may be of reflex origin, arising from nose, larynx, or vagal nerve endings of the lower airways, or it may be an allergic disease due to any of an endless number of allergens. Tracheal and bronchial nerve ends may be irritated and status asthmaticus caused by foreign body, tumor, bronchial rupture of the lymph nodes, etc. Therefore, in the absence of allergic signs and nasal disorder, bronchoscopy is indicated. Allergic asthma may be caused by the bacteria of chronic bronchitis. In these cases, a culture of bacteria may be taken and sensitivity determined. Autovaccine also may be produced and administered.

In both types of asthma the extremely thick, tenacious secretion may accumulate in the bronchial tree and cause atelectasis or dyspnea. In these cases therapeutic bronchoscopy is indicated.



# Pneumonia

Some readers may be surprised to find the subject of pneumonia included in this monograph. Indeed, does the bronchologist want to invade the territory of this characteristically internal disease? Does he really want to cure pneumonia through his bronchoscope? The answer is "Yes!" He wishes to invade and with his special instruments to cure some special forms of pneumonitis which are closely connected with a bronchial disease. Pneumonia interests the bronchologist in many respects: its prevention, diagnosis, duration and chronicity and its complications. The complications of pneumonia are discussed in Chapters 8, 9 and 12.

## Prevention

Pneumonitis evolves in many cases from a bronchial disease. Bronchial inflammation and infected bronchial secretions infect the alveoli. Bronchial stenosis hinders the cleaning of airways so that secretion accumulates distal to the stenosis. From an obstruction of the bronchi, first atelectasis results and then, by its infection, pneumonitis. It is obvious that a bronchial lesion, stenosis or obstruction must be removed in order to prevent the development of pneumonia.

**Pulmonary Atelectasis.** With regard to prevention, the bronchologist is first of all interested in pulmonary atelectasis. Bronchoscopy is indicated in any form of atelectasis. The author is aware of the question of reflex atelectasis, that some writers connect even obstructive atelectasis with the pulmonary musculature system. It is

and musculature.

However,

Actually, by removing obstructions and pneumonitis prevented. Space does not permit a type of atelectasis preceding pneumonitis, only those most important from the bronchologist's point of view will be mentioned.

**ATELECTASIS OF A NEWBORN INFANT** It is well known that this is caused by aspiration of amniotic fluid, meconium, vernix caseosa, or lanugo. Some degree of pulmonary airlessness existing for several days is physiologic but if it is extensive and lasts more than a week or two, intervention is indicated. Symptoms of atelectasis of the newborn caused by bronchial obstruction are as follows:

1 Progressive dyspnea with cyanosis, more pronounced after crying or other exertion. With oxygen insufflation it can be eliminated.

2 Suprasternal and epigastric indrawing, decreased thoracic movement on one or both sides, weakened respiration.

3 Rhonchi and local emphysematous areas.

4 Roentgenographic shadow extending to one or more lobes.

In these cases the newborn must be relieved of the obstructing material. First we attempt the simpler methods. Through the direct laryngoscope an aspiration tube with a rubber end may be introduced under fluoroscopic guidance and aspiration performed on the atelectatic side. Oxygen and warmth may be used, and the respiratory center stimulated.

If after this regimen bronchial obstruction does not disappear or the child is exhausted, bronchoscopy is urgently indicated, and the obstructing material must be aspirated under visualization.

Because of his exhaustion the state of the newborn does not improve instantly after this treatment. The obvious improvement generally occurs after 6 to 8 hours.

Atelectasis of the newborn infant involving a whole lung may be caused by a congenital web in the main bronchus or by stenosis from other causes. The congenital web may be pierced with the bronchoscope or with a probe through the tube, and thus atelectasis may be eliminated. These abnormalities are very rare.

**ATELECTASIS CAUSED BY THICK SECRETIONS** In atelectasis caused by thick secretion we have a peculiar situation. Previously the use of the bronchoscope was generally accepted in adults but not unanimously in infants and young children. Some writers thought bronchoscopic treatment inadvisable at this age because of its danger. These papers are surprising and create the doubt that a skilled bronchologist was at the writer's disposal.

The increasing use of the bronchoscope resulted in a most unexpected experience, here as in all human endeavor, knowledge arose wherever doors were opened wider. It was frequently observed that after simple diagnostic bronchoscopy atelectasis disappeared quickly and unexpectedly. This clarification came about even in children where atelectasis had lasted for weeks or months in spite of the usual treatment.

What is the usual treatment? It may be carbon dioxide insufflation, repeated slapping of the chest wall to make the child cough, postural

## PNEUMONIA

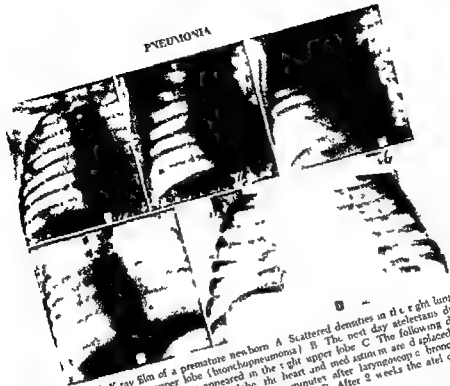


FIG 45 X ray film of a premature newborn A Scattered densities in the right lung especially in the upper lobe (bronchopneumonia) B The next day atelectasis due to obstruction of secretions appeared in the right upper lobe C The following day atelectasis extended to the middle lobe the heart and mediastinum are displaced to the right side D X ray film taken a few minutes after laryngoscopy a bronchial aspiration shows surprisingly prompt improvement E After 2 weeks the atelectasis has disappeared

drainage etc In infants such treatments are difficult or impossible to administer and they are certainly unsuccessful if the atelectasis lasts for a long period e.g. for months When these treatments were unsuccessful in a few cases diagnostic bronchoscopy was performed and surprisingly atelectasis disappeared Thick plugs of secretion were removed by suction Previously it had not been thought that these plugs might cause the atelectasis Thus a diagnostic procedure became a therapeutic method In most cases of this type atelectasis occurs during or after pneumonitis

In most cases of this type atelectasis occurs during or after pneumonitis pertussis measles and bronchial asthma (Fig 44) It appears also in cystic fibrosis of the pancreas Special attention must be called to the atelectasis in cases of poliomyelitis complicated by paralysis of the respiratory muscles The greatest danger for patients with poliomyelitis lying in a respirator is pulmonary atelectasis In these cases it is very important to prevent the development of an atelectasis from which pneumonia often develops Postural drainage may eliminate a large portion of the retained secretion but when atelectasis develops bronchoscopic aspiration or even a more radical method (see





FIG 46 X-ray films of a premature newborn After influenza, atelectasis appeared in the left lower lobe A, B The atelectatic lower lobe, instead of decreasing as is usual, increased in volume (drowned-lung or abscess type atelectasis), but the displacement of the heart and mediastinum and the clearer area inside the shadow at the cardiophrenic angle (A) are still unexplained, valvular emphysema in the left upper lobe must be considered Both atelectasis and emphysema are due to thick secretions The above mentioned clearer area B projected by the inflated lingula Two weeks after laryngoscopic aspiration, the previous changes had disappeared



FIG 47 X ray films showing pneumonia in the apical segment of the left lower lobe in a thirty five year-old man after appendectomy A Anteroposterior B Lateral

Repeated Aspirations in Chap 4) is indicated. Bronchoscopic aspiration has often proved to be a lifesaving procedure.

Atelectasis caused by thick secretion is frequently observed in poorly developed and undernourished infants or in children because of their underdeveloped respiratory musculature and insufficient expectoration (Figs 45 and 46). In adults it is more likely to be observed after thoracic or abdominal surgery when expectoration is hindered (Fig 47).

The following points are summarized from the literature.<sup>12</sup> In children with negative reaction to tuberculin test atelectasis cleared up within 10 days after the first bronchoscopy in 63 per cent and within 1 month in 20 per cent but in 17 per cent treatment was unsuccessful. The poorest results appeared in pertussis in spite of intensive treatment with sulfonamides, antibiotics and postural drainage in these cases atelectasis persisted sometimes for more than 5 weeks after bronchoscopy. In nontuberculous children 83 per cent of the atelectasis that failed to react to the usual conservative treatment disappeared within a month after bronchoscopic treatment.

In atelectasis if no other cause exists thick secretions must be considered. Regardless of its etiology atelectasis must be abolished as soon as possible. First the simpler treatments—postural drainage continued day and night, breathing exercises, expectorants—should be administered for removal of a plug. If no improvement is observed within a week bronchos-

suppuration it is a chief cause of bronchiectasis. From observations of a great number of cases it was stated that the same pulmonary parenchyma that had been atelectatic later became bronchiectatic. Furthermore it was also observed that the development of bronchiectasis was closely connected with the duration of the atelectasis: the longer the duration the worse the prognosis. It was stated that sometimes an atelectasis of 10 weeks duration caused irreversible bronchiectasis; after 6 months there was little hope and after a year expansion of an atelectasis could not be expected. However the author observed cases in which this statement was not verified (Figs 48 and 49). It is probable that a juvenile pulmonary disease is responsible for the unexplained etiology of bronchiectasis in an adult.

The use of the bronchoscope is not indicated for the treatment of influenza, other acute bronchial catarrh or inflammation, pneumonia developing from these diseases or an atypical perhaps virus type of pneumonia (Fig 50). These conditions must be treated with adequate internal medication. However if the bronchial inflammation is pathologically prolonged or if bronchial stenosis or atelectasis occurs bronchoscopy is in-



\* lateral fluoroscopic  
 image showing  
 a dense consolidation

regression of the previous changes after two bronchoscopic aspirations and lavage with penicillin and streptomycin solution



FIG. 49 Lateral x-ray film and bronchogram of a four year old boy. A Film showing a completely atelectatic middle lobe 2 years after pertussis. B Bronchogram showing the crowding, shortening and distortion of bronchi of the middle lobe (characteristic of deforming bronchitis and chronic lobar pneumonia). Note filling defect in middle lobe bronchus. C Fibrous debris 5 by 6 by 10 mm in size removed bronchoscopically from the middle lobe bronchus.



FIG. 50 X-ray films showing atelectasis of lingular inferior segment of a thirty five year old woman. A Anteroposterior. B Lateral. The atelectasis was due to swelling of the mucosa after an atypical (virus) pneumonia. The patient was treated and cured by internal medication. (From the collection of the Chevalier Jackson Clinic Philadelphia.)

perative In the great majority of these cases the cause may be revealed and eliminated only bronchoscopically

*Foreign bodies* frequently cause atelectasis and pneumonia This subject is discussed in detail in Chapter 12

*Rupture of tuberculous lymph nodes* will not be considered here as a tuberculous lesion but as a cause of a nontuberculous i.e. suppurative pneumonia Tuberculous cheesy debris represents a foreign body in the bronchus It induces pathologic mediastinal motion valvular emphysema atelectasis pneumothorax or even surprising combinations of these and through these purulent pneumonia may result (Figs 64-66)

### Diagnosis

Acute and chronic bronchial inflammations of interest to the bronchologist have been discussed Types of bronchial stenosis anatomic and functional factors and symptoms have also been mentioned The etiologic factors are summarized in the following outline (from Holinger and Andrews<sup>22</sup> with a few alterations)

- I Intrabronchial causes
  - A Endogenous
    - 1 Secretion
    - 2 Blood
    - 3 Broncholith
    - 4 Ruptured lymph node content
  - B Exogenous
    - 1 Foreign body
    - 2 Gastric content
- II Endobronchial causes
  - A Congenital web
  - B Nonspecific inflammation
    - 1 Edema
    - 2 Congestion
    - 3 Cicatricial stenosis
  - C Specific inflammation
    - 1 Tuberculosis
      - a Edema
      - b Congestion
      - c Granulation
      - d Ulcer
      - e Stenosis
    - 2 Syphilis
    - 3 Rhinoscleroma
    - 4 Leprosy

- D* Deformity of the walls
  - 1 Deviation
  - 2 Rotation
- E* Tumors
  - 1 Benign
  - 2 Malignant
- III Extrabronchial causes
  - A* Inflammation
    - 1 Enlarged lymph node
    - 2 Mediastinal abscess
    - 3 Disease of the vertebrae
  - B* Cysts
    - 1 Pulmonary
    - 2 Mediastinal
  - C* Emphysema
  - D* Tumors
    - 1 Mediastinal
    - 2 Pulmonary
    - 3 Esophageal
  - E* Cardiovascular
    - 1 Aneurysm
    - 2 Cardiac disease
      - a* Left auricular dilatation
      - b* Congenital hypertrophy
      - c* Anomalies
  - F* Esophageal foreign bodies

The general practitioner internist pneumologist pediatrician or roentgenologist may make a diagnosis of pneumonia but if a pulmonary unit (lung lobe segment) is involved and bronchial stenosis is suspected the supplying bronchus must be examined for the etiologic lesions. If pneumonia is cured with antibiotics and recurs at its initial location bronchoscopy is especially important. It is a great error to wait for repeated recurrences. The case of a forty five year-old male yields an example. A series of doctors observed and treated with antibiotics pneumonia of the patient's left lower lobe twenty four times within 2 years. His last physician awoke to the need for bronchoscopy and advised it. An adenoma 10 by 12 by 22 mm was removed from the left lower lobe bronchus (Fig 89). The patient was very fortunate to have survived his frequent attacks of pneumonia without developing irreversible bronchiectasis. This rule must be learned. Pneumonia repeated twice in the same location must arouse suspicion of disease in the supplying bronchus and indicates bronchoscopy.

It has been stated repeatedly that pneumonia involving a lobe or seg-

ment may be caused by a bronchial lesion. Suspicion of such a lesion is strengthened if the pneumonia becomes prolonged or if it is chronic. Another important rule is the following. In any case of lobar or segmental pneumonia lasting more than 2 weeks bronchoscopy is indicated; the indication is more imperative if the pneumonia is chronic. The cause that prevents recovery must be searched for in the bronchi.

### *Middle lobe Syndrome*

In this respect the most vulnerable unit of the lungs is the right middle lobe. Graham et al.<sup>7</sup> recognized the importance of this peculiar vulnerability and gave this lesion the general name *middle lobe syndrome*. They categorized by this term a condition of the right middle lobe resulting from compression of the lobar bronchus by hilar or peribronchial lymph nodes with secondary changes in the pulmonary parenchyma. Since their report in 1948 the term has been widely used by other writers for atelectasis or chronic pneumonitis limited to the middle lobe regardless of the etiology of the lesion, even cases without compression or occlusion of the bronchus were put in this category. Under this name various writers discuss the following diseases: atelectasis and chronic pneumonitis with secondary bronchiectasis; chronic abscesses with organizing pneumonitis; atelectasis and chronic pneumonitis secondary to tuberculous lymphadenitis; histoplasmosis; silicosis; carcinoma; adenopathy related to lower esophageal stricture; bronchial foreign body, etc.

The author has observed a series of cases of chronic middle lobe pneumonia due to thick, sometimes fibrinous secretion without adenopathy or bronchostenosis<sup>14</sup> (Figs. 48 and 49). The inflammation in these cases was not destructive and was treated very successfully bronchoscopically. However, one cannot give a simple anatomic explanation for these cases showing the peculiar vulnerability of the right middle lobe. Brock<sup>11</sup> gave as etiologic factors the length and narrowness of the middle lobe bronchus and its acute angle of origin from the stem bronchus and its closer relationship to lymph nodes that drain the right lower lobe as well as the middle lobe and other lobes. Observations by the author and other investigators suggest that functional factors (perhaps weaker aerodynamics of the middle lobe) may be another explanation of the peculiar vulnerability of the right middle lobe (see also Chap. 2).

### *Summary*

Pneumonia interests the bronchologist in regard to prevention, diagnosis, prolongation and chronicity, and complications.

With regard to prevention the bronchologist is interested in pulmonary atelectasis from which pneumonia may develop. Atelectasis may occur in the newborn because of aspiration of amniotic fluid, meconium, vernix

caseosa or lanugo or it may be due to congenital web especially when a whole lung is involved. At a later age it may be caused by many factors. Bronchoscopy is indicated in any case of atelectasis. Thus the normal lumen of a stenosed bronchus and ventilation of the distal parenchyma may be restored and development of pneumonia prevented.

In cases of pneumonia involving a pulmonary unit (lung lobe or segment) lesion of the supplying bronchus (lung lobe or segmental bronchus) is suspected and calls for urgent bronchoscopy when the pneumonia occurs twice in the same location. The suspicion of such a lesion is also strengthened if pneumonia is prolonged for more than 2 weeks and particularly if it is chronic. In these cases bronchoscopy is strongly indicated.

The right middle lobe is peculiarly vulnerable to atelectasis. The general term for this lesion is *middle lobe syndrome*. Brock gave as etiologic factors the length and narrowness of the middle lobe bronchus, its acute angle and the closeness of lymph nodes around it. Observations of the author and other investigators suggest that functional factors (perhaps weaker aerodynamics) may be another explanation for the peculiar vulnerability of this lobe.





## Pulmonary Abscess

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Pulmonary abscess arises from necrotizing pneumonia (Fig 51) and single or repeated inspiration of septic material. Hematogenous embolic abscesses also exist but according to recent research are rarer than bronchogenic ones.

In the majority of cases the septic material (pus blood tissue) is derived from the upper respiratory tract or from the mouth: puranasal sinusitis, dental caries, tonsillitis or adenoid or tonsil tissue aspirated during operation. These substances may be aspirated in sleep during unconsciousness or at the time of nasal pharyngeal or laryngeal operations or removal of teeth. During operations performed in the sitting position infection enters the lower lobes more frequently and the right middle lobe less frequently. After thoracic or abdominal operations respiration is weakened and expectoration is hindered. Because of the diminished phrenic respiration secretions accumulate in the lower lobes and may cause abscess formation. In sleep or after operation when the patient is lying in the recumbent position secretion accumulates in the apical segments of the lower lobes; in the lateral position it accumulates in the axillary subsegment and in the half lateral position in the posterior segment of the upper lobe (Fig 52). Therefore pulmonary abscesses appear particularly in these segments; they are caused by inspiration and their location is determined by the patient's position. Thus a pulmonary abscess is generally an *inspirational and positional disease*.<sup>1</sup> A pulmonary abscess may arise from an aspirated foreign body, a degenerating malignant tumor (simulating an abscess), a septic bronchial or alveolar cyst ruptured into or communicating with a bronchus or an infected echinococcus cyst. An abscess may be caused also by inflammation penetrating into the parenchyma from the neighboring organs (mediastinum, pleural cavity).

A pulmonary abscess frequently appears in the upper lobes. Formerly every process located in the upper lobes was considered to be of tuberculous pulmonary apical catarrhal origin and in the lower lobes to be an abscess. However it is a fact that a tuberculous pulmonary lesion is also an inspirational and positional disease as is pulmonary abscess.<sup>1</sup> Therefore the tuberculous primary focus (like an abscess) is mainly in the

axillary subsegments and posterior segments of the upper lobes (e.g., Asmann focus) or the lingular superior segment (Fig 53) and in the apical segments of the lower lobes (Fig 54)

Pulmonary abscess generally is a segmental disease. It is located in a segment and many times involves a total segment. In more severe cases it may extend to a whole lobe (Fig 51). In 75 per cent of the cases the abscess is solitary; two abscesses (in two lobes or bilaterally) are seldom observed. In the beginning pus in an abscess arising from pneumonia con-



FIG 51 X-ray films showing pneumonia extending to the whole left upper lobe. A Anteroposterior. B Lateral. The upper lobe necrotized and became a large abscess which later was lined with epithelium. C D Anteroposterior and lateral bronchograms respectively showing the cavity. Lobectomy was performed.

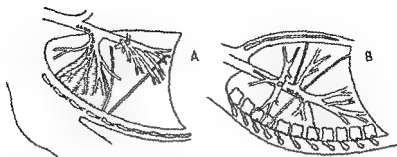


FIG. 52 Schematic drawing, showing the development of a pulmonary abscess. A The secretion enters the auxiliary subsegment of the posterior segment in lateral or left lateral position. B The secretion accumulates in the apical segments of the lower lobes when the patient is in the dorsal recumbent position. (From Brock 11)

tains pathogenic organisms. They are mostly staphylococci, streptococci, or Friedländer's bacillus. The open abscess sooner or later requires secondary infection. Spirochetes and fusiform bacilli occur chiefly in cases of dental origin.

**Signs and Symptoms.** The symptoms of an abscess are fever, irregular or intermittent pain, not always present but sometimes severe, cough, sputum slight at the beginning and later profuse, yellowish, greenish, and quite often putrid, sometimes bloody, leukocytosis with increased sedimentation rate, and malaise.

The x-ray appearance of an abscess before it ruptures into a bronchus and before it discharges pus is similar to that of pneumonia. After its rupture into a bronchus, air enters and occupies the upper portion of the cavity, and the x-ray shadow looks like that of a basket with a handle.



FIG. 53 X-ray film shows an abscess in the left lingular superior segment and empyema in the right pleural cavity of a three-year-old girl. A Anteroposterior. B Lateral.



FIG 54 Chest x rays of a two year old girl A B Lateral and anteroposterior films respectively showing huge abscess in the apical segment of the right lower lobe. Note that the shadow is similar to a basket with handle. The abscess cleared after bronchoscopic aspiration and postural drainage.

(Fig 54) The shadow of the handle represents the half circle of the upper margin of the cavity. The shadow of the basket's body represents the pus in the cavity. This *basket shadow* is very characteristic of a pulmonary abscess. Obviously its localization requires at least anteroposterior and lateral x ray films (Figs 53-56).

The bronchologist takes part in the prophylaxis, diagnosis and therapy of an abscess.

**Prophylaxis** Prevention was discussed in detail in Chapter 7 on Pneumonia. The bronchologist may abort the development of an abscess by abolishing atelectasis, thus preventing the development or prolongation of pneumonia. It is also self-evident that accumulated secretion must be aspirated from the bronchi after operations. Paranasal sinusitis, suppurating tonsillitis, and dental caries must also be treated.

**Diagnosis** In most cases of abscess, bronchoscopy is indicated. A diagnosis based only on symptoms, physical signs, and laboratory and x-ray examinations is uncertain and inadequate. The bronchologist, even in case with the most characteristic symptoms, may find stenosis, a foreign body, or tumor in the bronchus. The symptoms and the above-mentioned examinations do not yield conclusive information about these lesions. The bronchologist has not finished his work if in the visible bronchi he has found no other changes such as congestion, which is always present in the draining bronchus. In these cases, bronchography may yield further information. An accordion-like shadow of a bronchus caused by axial compression may prove to be a cyst or a benign tumor; truncation of a bronchus or of several bronchi suggests a malignant tumor (Fig 96). If the

abscess does not fill with simple bronchography aimed bronchography is indicated. With this method the cavity usually can be filled (Fig 55).

The configuration of the contrast shadow of a cavity may suggest important conclusions. In cases of pulmonary abscess the performance of cytologic, general and tuberculous bacteriologic examinations are necessary. They may present surprising data. According to the bacteriologic findings adequate antibiotics may be selected. This is especially important if a culture of pathogenic organisms taken from the secretion is made. A pure culture may be expected only from sterile aspirated secretion obtained through the bronchoscope. A pure culture of pathogenic bacteria is an important diagnostic sign. It proves the presence of *fresh primary abscess*.

When the slightest suspicion of a malignant tumor arises in a patient over forty e.g. if the abscess was preceded by atelectasis or pneumonia

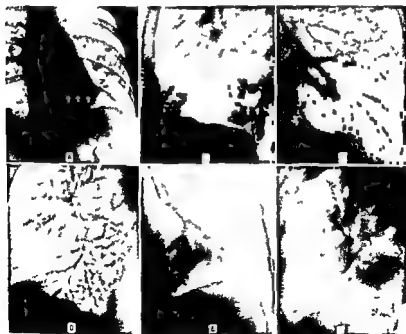


FIG. 55. X-ray films showing an abscess in the posterior basal segment of the left lower lobe of a thirty-five-year-old woman. A. Anteroposterior. B. Lateral. C. Two years later the abscess cavity was lined with epithelium but did not fill when routine bronchography was performed. E, F. Aimed bronchograms showing the large cavity. Lobectomy was performed.

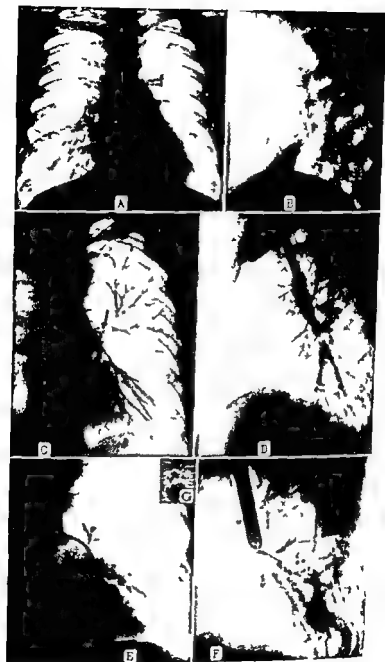


FIG 56 Chest x rays and bronchograms of a twenty three-year old man who had

recurring in the same location and no tumor tissue was noted in the bronchial orifice. A suitable delicate instrument may be inserted into the depth of the draining bronchus to obtain tissue for histologic examination. This procedure often results in an unexpected histologic diagnosis. The question of a "blind" biopsy will be discussed in Chapter 11, on Bronchial Tumors.

The following case report demonstrates one example of a surprising result of this diagnostic procedure.

**CASE 7** A twenty three year old male had an occasional hemoptysis from 1948 to 1953. In 1953 the continued bleeding caused severe anemia and he received repeated blood transfusions. A bronchogram was made on the left (Fig. 56C and D) and the apical segment of the left lower lobe did not fill. (This was done at another hospital. Regularly bronchography is never performed before bronchoscopy.) In June 1953 bronchoscopy was performed and the left bronchus discharged fresh blood. With aimed bronchography a small walnut sized cavity filled in the lower portion of the left apical segment (Fig. 56E and F). The patient coughed up thick contrast fluid Joduron B (propyl iodine) and with it a small (3 mm in diameter) metal foreign body (Fig. 56G). Since then hemoptysis has never returned. Three months later control aimed bronchography showed the disappearance of the abscess.

A foreign body in the apical segment of the left lower lobe caused an abscess with severe hemoptysis for 5 years. The abscess was revealed by aimed bronchography. In this case aimed bronchography was not only a correctly applied diagnostic procedure but also a therapeutic maneuver.

**Therapy** Generally an early abscess is easily cured. At first internal medication is administered—e.g., light diet, plenty of vitamins, symptomatic treatment, postural drainage, blood transfusions, sulfonamides, antibiotics—penicillin, streptomycin or others—and perhaps a vaccine. Antibiotics and postural drainage are the most important therapies. The effect of antibiotics is well known. In postural drainage the patient is placed continually if possible, in such a position that drainage from the bronchus is aided by gravity, the trachea sloping downward and toward the mouth. For example, if the abscess is located in the right axillary subsegment the patient should lie on his left side and the foot of his bed should be elevated. When the abscess is in the apical segment of a lower lobe the patient should be placed in a prone position with the foot of the bed elevated. In this position pus is continually discharged and the patient's condition usually improves within a few days.

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bronchus) E, F. Antero-posterior and lateral aimed bronchograms respectively showing a small walnut sized abscess in the apical segment of the left lower lobe. The patient discharged with the contrast material a small metal foreign body 3 mm in diameter. Hemoptysis has never returned.



If in an infant or a child there is no suspicion of a foreign body and the patient improves rapidly with the above treatment, bronchoscopy can be omitted. However, bronchoscopy is indicated if no improvement is observed within several days (Fig 54). This is equally true for adults. However, in older children and adults it is advisable to perform bronchoscopy even when there is improvement in the patient's condition. In this way, repeated recurrence of pneumonia or an abscess may be avoided. It must be remembered that the patient will improve under the above treatment even though the etiologic factor of an abscess is located in the supplying bronchus.

Bronchoscopic treatment of an abscess in some cases begins with elimination of the cause. In a great majority of the cases an abscess caused by a foreign body will be completely and rapidly cured with the removal of the foreign body. When no foreign body or other lesion, except the associated mucosal inflammation, is found in the bronchus, a flexible aspirating tube may be inserted into the draining bronchus and the pus aspirated. By this aimed aspiration secretions may be extracted from the smaller peripheral bronchi as well as from those that deter evacuation of the abscess. After each aspiration the proper antibiotics may be instilled into the abscess through the suction tube. The patient is requested to breathe deeply, this aspirates the antibiotic solution into the cavity. Five milliliters of a solution containing 40 000 units of penicillin or 0.1 Gm of streptomycin is recommended. These antibiotics may be used simultaneously. Bronchoscopic treatments are usually repeated once or twice a week. If the aspirating tube enters the abscess cavity, the effect of the treatment will be more pronounced. Penetration into the cavity may be achieved also with a Metras' catheter.

Serial bronchoscopic treatment is acceptable only in cases of acute abscesses. Usually, bronchoscopic treatment is unsuitable for abscesses of more than 8 weeks' duration. However, if bronchoscopic treatment is begun early and within a few weeks has not been successful, the patient should be placed in the hands of a surgeon, who may then perform a Monaldi drainage or pneumonotomy. In chronic or epithelialized abscesses, depending on the extent of the abscess, the involved segment or lobe may require resection (Fig 51).

Prior to such surgical procedures the bronchologist may perform bronchial aspiration and lavage to diminish the secretion. Sometimes only by this treatment can debilitated patients be sufficiently improved to permit surgery. Postoperatively, the bronchologist aspirates the retained secretion, eliminates atelectasis or removes sutures extruded into the amputated bronchus.

*Summary*

Pulmonary abscess is characteristically a segmental disease. Generally, the lesion is located in a segment, frequently it involves a total segment. Being an inspirational and positional disease, pulmonary abscess appears typically in the axillary (lateral) subsegments or in the posterior segment of the upper lobes and apical segment of the lower lobes, because secretions gravitate into these segments when the patient is in the lateral or dorsal recumbent positions, respectively.

The bronchologist takes part in prophylaxis, diagnosis, and therapy. A pulmonary abscess develops most frequently from necrotizing pneumonia. Diagnosis of a pulmonary abscess based only on symptoms, physical signs, laboratory and x-ray findings is uncertain and inadequate. The bronchologist, even in cases with the most characteristic symptoms and signs of an abscess, may find a foreign body, tumor, or other stenosis in the supplying bronchus. Therefore bronchoscopy is indicated in almost every case of pulmonary abscess.

Bacteriologic examination and bronchography may yield further information even if bronchoscopy shows only the typical mucosal congestion in the draining bronchus. A pure culture of pathogenic bacteria indicates a fresh, primary abscess, and bronchography reveals the signs of cyst or extrabronchial benign or malignant tumor. In cases with uncertain diagnosis of an abscess, aimed bronchography is the proper method by which the cavity usually can be revealed.

Generally, an early abscess is easily cured with proper therapy including diet, vitamins, postural drainage, blood transfusion, antibiotics, and sometimes vaccine. However, if no improvement is observed within a few days, bronchoscopy is indicated for both diagnostic and therapeutic reasons. Bronchoscopic treatment is unsuitable for abscesses of more than 8 weeks' duration.



# Bronchiectasis

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*Bronchiectasis means dilatation of the bronchi. But these bronchiectatic dilatations do not appear as expansions or distentions of major bronchi the latter are caused rather by deforming bronchitis. Typical bronchiectatic dilatations are always located at the distal end of the minor bronchi. The quaternary or smaller branchings end in cystic cylindrical sacular or clubbed dilatations (Fig 57). Generally bronchi beyond the subsegmental cannot be viewed through a bronchoscope. Thus bronchoscopically the lesion itself is never visible.*

## *Hereditary or Congenital Bronchiectasis*

Bronchiectasis may be *hereditary* or *acquired*. Some decades ago physicians believed that the great majority of cases of bronchiectasis were hereditary or congenital. Furthermore this statement was supported by many different observations. For example in investigations and examinations of hereditary diseases in twins bronchiectasis was found in both of them or in several members of the family. The impression that the disease was congenital was supported by cysts which appeared also in other organs (kidney, liver, spleen etc.) and then by bronchiectasis in accessory lobes and by bilateral bronchiectasis.

All the evidence appeared very plausible. But later clinical observations numerous surgical specimens and many experiments opposed this supposition. Today only the cystic lung may be considered a congenital disease. In a cystic lung minor or major cysts are scattered throughout (Fig 58). Sometimes a whole lung is filled with these cysts. The scattered cysts communicate through very thin channels with the bronchi and are surrounded by intact lung parenchyma. However it cannot be concluded that all bronchiectases surrounded by healthy parenchyma are congenital. No doubt in the great majority of cases bronchiectasis is enclosed in fibrotic suppurating tissues but it will be seen later that the pulmonary parenchyma embracing bronchiectasis is not necessarily fibrotic or suppurated especially in childhood. The lungs are developing until the child attains the age of seven to fourteen years. During this growth they



FIG 57 Schematic drawing showing saccular bronchiectasis in the inferior segment of the lingula and in the lower lobe



FIG 58 Bronchograms of a twelve year old girl showing scattered cysts in the right lung. The channels communicating with the bronchi are very narrow. Congenital bronchiectasis was the diagnosis

may surround ectatic bronchi with healthy pulmonary tissue substituting it for the destroyed parenchyma. Bronchiectasis frequently arises in childhood but it does not often become a serious disease until adulthood. Thus an adult may carry bronchiectasis surrounded by intact parenchyma from his childhood. Finally, certain acquired bronchiectases have been observed that were undoubtedly caused by a foreign body and surrounded by intact lung tissue.

In many countries except for tuberculosis bronchiectasis is the most frequently occurring pulmonary disease. If the disease were congenital in most cases it would have been noted frequently at the autopsies of infants. Certain parts of autopsies may be performed with negligence however after the pathologist's attention was called to this matter even the most precisely performed autopsies showed no evidence of bronchiectasis. Many researchers focused their attention in this direction. According to the theoretical suppositions congenital bronchiectasis should occur in connection with other abnormalities but bronchiectasis was not found in a single case out of fifty corrosion preparations of bronchial trees of monstrosities and newborn infants with severe congenital abnormalities.

#### *Acquired Bronchiectasis*

This may be primary or secondary. The primary acquired bronchiectasis (Chap. 8) develops as a result of chronic bronchitis and deforming bronchitis by weakening the bronchial wall.

The causes of secondary acquired bronchiectasis have less connection with the bronchial wall. Such bronchiectases arise near destroyed shrunk pulmonary tissue as a sequel to compensatory emphysema but may occur also in emphysematous parenchyma. Some writers speak directly of "bronchial emphysema" as "Pleural callus may affect traction and dilatation of the bronchial wall and so on."

Bronchiectasis has been produced experimentally for the study of its genesis. Bronchial arteries (trophic vessels) and nerves were cut around the bronchial wall. In these experiments the writers thought they had found etiologic evidence in trophic disturbance. Bronchiectasis was produced also by artificial occlusion of a bronchus. Some writers state that occlusion and the consequent atelectasis alone is insufficient to produce bronchiectasis. Others emphasize that atelectasis alone is insufficient and supuration must be present in the occluded parenchyma. Numerous writers support the view that supuration together with weakening of the bronchial wall are insufficient to produce bronchiectasis because normally 1 atm of pressure is unable to dilate bronchi persistently therefore the expansive force of retained secretion is also necessary (Figs 59 and 60). In cases of incompletely occluded plugs or stenosis

some writers <sup>60</sup> believe that a valvular action is present and state that distention by air is responsible for the development of bronchiectasis (Fig 61) This conclusion is supported in animal experiments by the fact that bronchiectasis can be produced by valvular obstruction of a bronchus Cough may be a factor in the expansive effect, but cough itself does



FIG. 59 A forty six year old man aspirated the mouth end of a cigarette holder during an epileptic seizure. A Arrow indicates where it lodged in the left main bronchus for 4 years and obstructed the upper lobe bronchus while the lower lobe breathed through the lumen of the holder. Thus the upper lobe was under pressure of the stagnated foreign body in the upper lobe which in this case was free from pressure and also remained free of infection. (B) Bronchogram in E was aimed because the lingula remained empty with normal bronchography. C The removed foreign body.

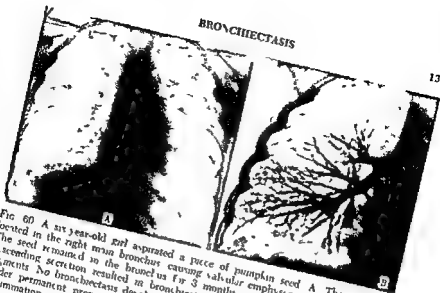


FIG. 60 A six year-old girl aspirated a piece of pumpkin seed. A The seed was located in the right main bronchus causing valvular emphysema in the right lung. The seed remained in the bronchus for 3 months causing inflammation. B The descending secretion resulted in bronchiectasis in the medial and posterior hilar segments. No bronchiectasis developed in the other portion of the lung which was under permanent pressure, but free of inflammation. Only the pressure plus the inflammation caused bronchiectasis.

not stretch the bronchial wall. During cough the bronchial system is under strong inner pressure but simultaneously just as strong in outer thoracic pressure. It is similar to a closed balloon that is under equal pressure from outside and inside. Neither the bronchi nor the balloon will expand with these combined pressures. Cough acts only as an intermediate effect when valvular action is present in a bronchus. In this case the stronger pressure of air persists after cough has ceased because the air is trapped in distal bronchi by the valvular action. This pressure then expands effectively.

As stated a weakened bronchial wall represents one of the conditions responsible for the development of bronchiectasis. In the bronchial wall smooth muscle and elastic fiber systems connect the cartilages giving solidity and elasticity. These elements in the wall of the ectatic bronchi are mainly destroyed. The dissolution of elastic fibers and thus the production of bronchiectasis were demonstrated in rats by the administration of carbon tetrachloride<sup>10</sup> and of urethane.<sup>12</sup> In pneumococcal fibrosis the elastic fibers are destroyed and bronchiectasis is also produced. In the opinion of certain Russian writers dysfunction of the autonomic nervous system has an effect in destroying elastic fibers and in producing bronchiectasis.

To recapitulate bronchiectasis arises first from destruction of bronchial elastic and smooth muscle elements. In this destruction the most important part is played by purulent inflammation of the bronchial wall. A similar





FIG 61 A B Anteroposterior and lateral bronchograms respectively showing a foreign body in the right lower lobe bronchus (see arrows) and bronchiectasis in the lower lobe. The foreign body (a piece of plum pit) was located in the right lower lobe bronchus above the basal branching. C It caused bronchiectasis in the intact parenchyma of the apical segment of the lower lobe probably by valvular action. D It also caused bronchiectasis in the basal segment surrounded by fibrotic tissues because of occlusion. Neither routine x ray examinations nor bronchoscopy detected the foreign body; it was found in the surgical specimen.

effect may be produced by certain chemicals or perhaps by dysfunctions of the nervous system. Dilatation itself is increased by the expansive effect of pus stagnating behind the stenosis and by occasional valvular actions.

Bronchiectasis occurs more frequently in the lower lobes than in the middle lobe and lingula and less frequently in the right upper and the left truncated lobes. It may affect a whole lung, a lobe, or a segment. On one side it may occupy two lobes or several segments. Its simultaneous appearance in the left basal segments and in the lingula is frequent (Fig 57). Bilateral bronchiectasis also is frequently observed.

Purulent bronchiectasis is usually a disease of adulthood, but with more exact diagnostic methods it may frequently be observed in childhood.

It has been mentioned that in the majority of cases bronchiectasis is

accompanied by profuse suppuration but this is not a rule symptomless bronchiectasis also exists When bronchial stenosis ceases to exist and cavities become completely evacuated in fortunate cases production of pus stops Bronchiectasis in the right upper and left truncated lobe is favorable because pus can be continually discharged and the inflammation then tends to heal Some types of bronchiectasis may remain hidden throughout an entire lifetime and therefore cannot be considered as a real disease The infected purulent bleeding bronchiectasis represents a real disease i.e. a severe disease On this basis two types of bronchiectasis may be considered a symptomless form and a "bronchiectatic disease"

**Signs and Symptoms** In "bronchiectatic disease" the author does not believe that the differentiation between a "dry" initial and a "wet" later period is correct Acquired bronchiectasis in the majority of cases is a sequel of bronchial and pulmonary suppuration When the cause of the suppuration (foreign body ruptured content of a lymph node or benign bronchial tumor etc.) is eliminated the bronchial cavities *exsiccate* Suppuration also may be excited with bronchial aspiration or lavage Later let us say at the time of the first so called "common cold" cavities will be newly infected and become "wet" Thus symptoms of bronchiectasis first simulate those of chronic bronchitis and during the progressive stages of the process conform with the symptoms of deforming bronchitis later the "dry" and wet periods may alternate

Bronchiectasis without suppuration may also be a disease and often not a harmless one Varices may be present in the thin walled "dry" cavities From these very severe bleeding may arise Repeated bleedings are more frequent and are more dangerous in bronchiectatic cases than pulmonary hemorrhage in tuberculosis or in carcinoma The most frequent and most severe episodes of hemoptysis originate from "dry" bronchiectasis of the right upper and left truncated lobes

Many degrees of bronchiectatic suppuration exist The sputum may be mucopurulent for a long period in scant quantities it may be more definitely purulent in a moderate amount from 10 to 20 ml per day but the secretion of so called "suppurative bronchiectasis" containing several large dilatations is white grayish yellowish or greenish thick and tenacious sometimes foul and putrid It may amount to a total of 500 ml daily This sputum left in a glass separates into the well known three layers the upper layer foamy the middle grayish yellowish or greenish and slightly transparent and the bottom purulent debris Severe infected bronchiectasis is recognizable from the cough of the patient The patient brings up voluminous amounts of sputum from large cavities (septic tanks) with a hollowish sound

In the vast majority of cases the bacterial content of the bronchiectatic pus is mixed

Fever appears only when acute inflammation is superimposed on the chronic bronchiectatic process. In these cases other signs of acute inflammation are present, increased sedimentation rate, increased leukocytes, with a primary shift to the left, etc. Agammaglobulinemia is associated with high incidence of respiratory tract disease including bronchiectasis, therefore every patient with bronchiectasis should be examined for globulin deficiency.

A ray signs of bronchiectasis in milder cases are indistinct, usually, only a few bunchy or mottled densities can be seen. Sometimes, especially in cases of bronchiectasis surrounded with almost normal parenchyma, bronchiectasis cannot be suspected even on examination of the routine x ray films; thus negative x ray findings do not exclude the presence of bronchiectasis (Fig. 62). In pronounced cases x ray symptoms are fairly characteristic. The bronchiectatic pulmonary tissues are generally shrunken, and inside these tissues the cavities are filled with air or pus.

Thus by x ray examination both the signs of shrinkage and the shadow of the involved pulmonary tissue are observed.

The shrunken pulmonary tissue pulls the neighboring lobes, segments or mediastinal organs toward itself. It can be noted by fluoroscopic examination or on films by the dislocation of one or another portion of the middle shadow, and in some cases by the dislocation of the linear shadow of the interlobar fissure. Dislocation of lobes or segments is best observed on bronchograms by dislocation of the corresponding bronchi (Fig. 63C).

The affected portion of lung projects as a massive shadow or scattered aerated spots according to the contents of the cavities, that is, according to whether they are filled with air or pus. The shadow is also massive if thick airless or cicatricial tissue embraces the cavities in which case aerated cavities are invisible. As we have seen, bronchiectases are more frequently caused by disease of a major bronchus and located in the minor bronchi of the distal parenchyma (Figs. 59, 60, and 63). Therefore, generally, bronchiectatic disease of a pulmonary unit (lung lobe, or segment) must be anticipated and the shadow of the shrunken pulmonary tissue will involve a lung lobe, or segment. For the best evaluation two x ray films exposed in sagittal and frontal projections are necessary. In atelectasis in the lower lobes, e.g., especially in the left side, the shrunken lobe hides itself behind the heart shadow on anteroposterior films and is visible only on lateral films. Cavities are demonstrable in plain films in many cases. The shadow of a cystic lung or portion of a lung is similar to a honeycomb.

The bronchologist is concerned in bronchiectasis with prophylaxis, diagnosis, and therapy.

**Prophylaxis.** One of the most important functions of the bronchologist is to prevent it. It was previously stated that

bronchiectasis is caused by stenosis and inflammation of a bronchus, which produces inflammation in the distal bronchi and pulmonary parenchyma. However, chronic inflammation may also appear without stenosis, as discussed in relation to chronic bronchitis and deforming bronchitis in Chapter II. The bronchologist therefore, in cases of bronchiectasis or

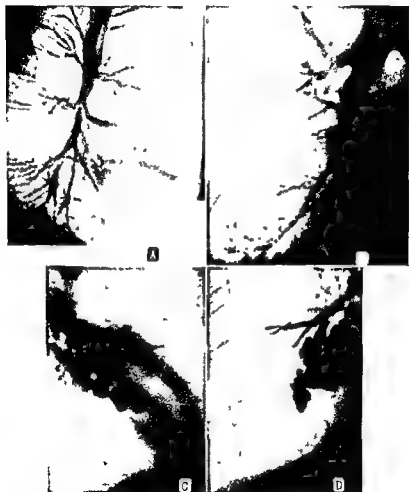


FIG. 62 A forty six year old man with frequent respiratory infections complicated many times by pneumonia whose x ray showed only very slight density in the region of the right middle lobe. A B Normal bronchograms showed substantial stenosis in the orifice of the right middle lobe bronchus which was filled only for a short distance. C D Aimed bronchography revealed extensive bronchiectasis in the medial segment of the middle lobe.

suspected bronchiectasis must think of inflammation in the upper respiratory tract (nose paranasal sinuses tonsils nasopharynx) Paranasal sinusitis may be seen in 50 per cent of the cases of bronchiectasis of unexplained origin. The effect of dust and chemicals must of course be considered too. It has been stated previously that bronchiectasis may be prevented by the cure of diseases of the upper respiratory organs and of chronic and deforming bronchitis. These treatments thus become prophylactic of bronchiectasis.

In bronchial stenosis no matter what its etiology pulmonary suppuration (abscess pyosclerosis bronchiectasis) is certain to develop in the distal portion of the lung. Almost every etiologic factor was noted in Chapter 7. Each of these may cause bronchiectasis. If the lesion can be eliminated and with it the bronchial stenosis bronchiectasis may be prevented. In Chapter 7 on Pneumonia we saw that atelectasis of an infant caused by inspiration of meconium or vernix caseosa can be eliminated. The same can be done in atelectasis of young children caused by thick secretion in pertussis measles or pneumonia a foreign body and the content of a ruptured tuberculous lymph node can also be removed. Intra-bronchial benign tumors may be removed in time. All these preventive measures can be and are being taken when the possibility of these causative factors is kept in mind!

Bronchoscopy must be performed in patients who persistently cough without any evident reason as in persons who are not heavy smokers in patients who suffer from chronic bronchitis hemoptysis or pulmonary atelectasis in cases of prolonged pneumonia or in recurrent pneumonia in the same site and in cases with positive Holzkecht's sign. If we are going to reduce the number of cases of bronchiectasis which occur in great numbers throughout the world bronchoscopy must be performed far more frequently not only in adults but just as often in infants and children. Naturally certain prerequisites must be established if such a program is to be created. These have not been established in most countries. The first difficulty is that the bronchologist whose basic specialty is pneumology generally does not perform bronchoscopy in infants and young children and furthermore relatively few laryngobronchologists are sufficiently skilled and dare to perform bronchoscopy in infants or young children. The second difficulty is that many pediatricians have not recognized the benefit of this prophylaxis.

Near the great medical centers in the United States where the prerequisites have been established the occurrence of the bronchiectasis appears to have been greatly reduced in the last few years.

**Diagnosis.** The diagnostic work of a bronchologist is at least as important as bronchoscopic prophylaxis. The diagnostic work may be divided into two parts: diagnosis of bronchiectasis and the search for a cause of

the bronchiectasis. Naming these two in this order is correct from the diagnostic point of view but from the bronchologist's point of view it is more appropriate to reverse the order because the bronchologist in most cases first discovers the etiologic lesion in the bronchus if it is present and only after this does he make a diagnosis of bronchiectasis. This procedure is natural because first the cause (foreign body, tumor, etc.) appears in the bronchus and then its consequence (bronchiectasis) beyond it.

A few observations must be made on the detection of the cause of bronchiectasis. The bronchologist performs bronchoscopy in cases with symptoms of long existing cough, atelectasis, hemoptysis, profuse expectoration of pus, positive Holzknöchel's sign, and in cases of prolonged or recurrent pneumonia, opaque foreign body, etc. If he finds a thick plug of secretion, granulation tissue, or cheesy debris, benign or malignant tumor, foreign body, cicatricial stenosis, then he has diagnosed the primary disease, but the condition of the distal parenchyma is still unknown. From the quality—thick, creamlike, tenacious, perhaps putrid—and quantity of the secretion, well based conclusions may be drawn regarding the cause of the bronchiectasis. However, since a precise diagnosis of bronchiectasis is possible only if the ectasies are shown in bronchograms, bronchiography is an integral part of the diagnosis. Therefore this procedure is considered very important, and if the involved or suspected portion of a lung in spite of careful aspiration of secretion remains empty or inadequately filled on a simple bronchogram, then, with aimed bronchiography, a contrast material is injected into the unfilled portion of the lung to complete the diagnosis (Chap. 4, Figs. 59, 62, and 67).

Sometimes the bronchologist does not see the etiologic change in the bronchus, but it still may be present somewhere in the lung. Again, it may be there but perhaps in a place that is invisible to the bronchologist or his view may be obstructed by excessive suppuration, which prevents the use of precise instruments, e.g., telescopes. I have observed some cases in which a foreign body that could not be detected bronchoscopically was found in the surgical specimen (Fig. 61).

Cases also exist in which the etiologic factors cannot be found because they have already disappeared from the bronchus. In these cases, cheesy debris or molded foreign body might have been expectorated after the development of the irreversible bronchiectasis.

Naturally, the cause of bronchiectasis cannot be found or even guessed in every single case. The bronchologist may observe only the characteristic and excessive secretion, congested mucosa, thickened spurs, and stenosis caused by the previous lesions. It has been established<sup>23</sup> that these mucosal swellings and stenoses are not due only to a simple chronic inflammation but also frequently to extensive hypertrophy and dilatation

of acini of the bronchial glands. These changes may represent a special form of chronic bronchitis. It is more probable that the hypertrophy is caused by the irritative effect of the inflammation and the dilatation by the retained secretion (Chap. 6). It is also possible that these changes are one of the etiologic factors of deformity of the bronchial wall and of narrowing of the bronchial lumens. It cannot be ignored that this glandular hypertrophy also predisposes to the development of bronchiectasis.

Cases still exist in which bronchoscopy does not reveal the cause of bronchiectasis, however, the number of these negative cases is continually lessening. The work of a bronchologist is useful even in these negative cases. With or without making the patient cough he observes the bronchus which discharges the secretion. In this way he may localize the lesion, or at least he may obtain a good impression as to which portion of the lung must be mapped by contrast filling for determination or exclusion of bronchiectasis. The chest surgeon needs this accurate localization because he wants to remove completely only the affected portion.

The bronchologist always sends secretions which were aspirated under sterile procedures for bacteriologic study, first, as a basis for a choice of antibiotic treatment, and, if the slightest suspicion of tuberculosis arises then for a smear and culture.

**Therapy.** In childhood after removal of an obstructing plug bronchiectasis may heal (Figs. 63C and E). These healing processes may be favorably assisted by bronchial aspiration and lavage with proper antibiotics.

The bronchologist's work is less important in the therapy of an already developed irreversible bronchiectasis, since he is unable to cure this type of disease. In these cases the condition of a patient may be improved by bronchial aspiration and lavage, sputum may lose its odor, decrease in amount or even cease, but sooner or later in almost every case, after therapy is discontinued the symptoms recur. Therefore, this treatment is used only if a case is unsuitable for surgery, for example, if the patient is too debilitated or too old, if operation is contraindicated because of a cardiac condition or other severe symptomatic disease, or if the patient refuses to accept operation.

Bronchoscopic therapy, naturally, must be complemented by the usual internal medication, good nutrition, rest, vitamins, aerosol, postural drainage, etc.

Bronchoscopic aspiration and lavage is of great importance in preparation for surgery. Prior to operation the amount of secretion may be markedly decreased or abolished. This treatment decreases the danger of suffocation and aspiration of infection to the opposite side and thus reduces the risk in surgery.

The surgeon removes the affected pulmonary parenchyma with resection



FIG. 63 A five-year-old boy aspirated the cartridge of a 6-mm bullet that was lodged in the right stem bronchus for 3 weeks and in the lower lobe bronchus for another 2 weeks. The cartridge was pushed down by unsuccessful bronchoscopy. A B The middle lobe was occluded and atelectatic for 3 weeks and the lower lobe for 5 weeks. C Deforming bronchitis developed in the middle lobe and pronounced bronchiectasis developed in the lower lobe. Dislocation of the right upper lobar branchings (C) demonstrates clearly the shrinkage of the middle and lower lobes. D The removed foreign body. X-ray films were taken and bronchograms made 1 year after the foreign body was removed. ■ Bronchogram showing absence of the bronchiectasis and normal appearance of the bronchial tree.



of a lung lobe, or segment. In irreversible bronchiectasis, pulmonary resection must be performed promptly even in a young child. Surgery should not be delayed until the resistance of a patient decreases or until cachexia ensues or amyloidosis threatens.

The bronchologist also uses his knowledge *postoperatively*. During the first postoperative hours or days, he aspirates accumulated secretions from the airway of the patient who is unable to expectorate, thus removing the threat of atelectasis or suffocation. Later, he treats bronchial fistulas which may occur or a consequent abscess. Lastly, he may remove sutures or ties extruded into the lumen of a resected bronchus, these represent foreign bodies and cause suppuration, granulation, and sometimes hemoptysis.

### Summary

Bronchiectasis may be hereditary or congenital, or acquired. Some decades ago physicians believed that in most cases bronchiectasis was hereditary or congenital. Today only the cystic lung may be considered of this origin. Acquired bronchiectasis may appear as primary and as secondary. Primary acquired bronchiectasis develops as a result of chronic and deforming bronchitis by weakening of the bronchial wall. Some writers have concluded that mechanical factors (inner pressure, valvular action) also play a part. Secondary acquired bronchiectasis is caused by extrabronchial shrinking processes.

Bronchiectasis may be symptomless or a severe disease. In its course "dry" and "wet" periods may alternate. The most frequent and severe episodes of hemoptysis originate from "dry" bronchiectasis of the right upper and left truncated (upper division) lobes, because varices are often present in the thin walled dry cavities.

By x-ray examinations the signs of shrinkage and the shadow of the involved pulmonary tissues are observed. The bronchiectatic cavities can be seen in planigrams and more precisely in bronchograms or in cases of "dead tree effect" in aimed bronchograms.

Bronchiectasis must be considered from the following symptoms:

1. Long persisting cough. The amount of sputum is not always characteristic. However, in most cases it is voluminous, especially in the morning since it accumulates during the night.

2. A diagnosis can almost certainly be made if pus is brought up in large amounts with strong coughing and with a hollow sound from the patient's lung. In these cases bronchiectatic disease is extensive and severe, the patient is often dyspneic.

3. Bronchiectasis must be considered in cases of severe hemoptysis with or without purulent expectoration.

4. The above symptoms are supported by cavernous or massive x-ray

shadows indicating a shrinking process and extending to a lung lobe or segment

5 In more severe cases the patient may have clubbing of the fingers

6 Fever accelerated sedimentation rate high leukocyte count shift to the left of the blood picture all indicate an acute inflammation and aggravation of the chronic disease

Bronchiectasis may be prevented by curing diseases of the upper respiratory organs or by curing chronic or deforming bronchitis. If pulmonary suppuration and with it bronchial stenosis can be eliminated bronchiectasis may be prevented. Atelectasis of infants and young children can be eliminated and other preventive measures can be taken. If the great number of cases of bronchiectasis now occurring throughout the world is to be reduced bronchoscopy will have to be used far more extensively than it is.

The diagnostic work of a bronchologist extends to the demonstration of and the search for a cause of the bronchiectasis. *Demonstration* refers to the bronchoscopic routine bronchographic and aimed bronchographic signs. Cause of a bronchiectasis may be found in nasal or pharyngeal disorders and/or in chronic and deforming bronchitis or any kind of bronchial stenosis. However a few cases still remain in which the bronchologist is unable to reveal the cause of bronchiectasis.

The bronchologist cannot cure an already developed bronchiectasis. Generally his therapy may be successful only in children. However in cases unsuitable for surgery bronchoscopic aspiration and lavage with proper antibiotics may improve the condition of the patient.



# Pulmonary Tuberculosis and Mycosis

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## TUBERCULOSIS

Before 1930 bronchoscopy was performed only sporadically on patients with pulmonary tuberculosis. Phthisiologists expressed the opinion that bronchoscopy did harm to the pulmonary process; therefore they were afraid of the procedure and would not agree to use it for examination. After 1930 improvement in bronchoscopic technique brought about alterations in this field as well as others. Phthisiologists learned that bronchoscopy is a harmless procedure in the hands of an experienced endoscopist even in tuberculous patients and that its advantages are much greater than its hazards. They realized soon that recognition of a tuberculous tracheitis or bronchitis was very important in the therapy of pulmonary tuberculosis. *Bronchial tuberculosis is recognizable only bronchoscopically.*

At the beginning of this period tuberculous lesions of the bronchi were generally treated through bronchoscopy. Since the discovery of effective antibiotics and other chemotherapeutic agents such as streptomycin, PAS (para-aminosalicylic acid), INH (isoniazid), bronchoscopic therapy has lost much of its importance, but it is still worthwhile.

Thus, while the importance of bronchoscopic therapy has decreased, its diagnostic value continues to increase. Very significant principles on the relation of pulmonary tuberculosis to bronchial tuberculosis have been stated as follows:

1. Lesions of the pulmonary parenchyma affect the mucosa of the draining bronchus.
2. The character of a lesion of the bronchial mucosa is similar to that in the lung.
3. The primary lesions (in the parenchyma or lymph nodes) extend in continuity to the bronchus and may reach a visible portion of the airway.
4. Bronchial lesions signalize improvement or aggravation of the pulmonary lesion with similar types of changes.

5 Therefore, in certain cases, the character or course of the pulmonary disease may be studied bronchoscopically

These principles represent examples of what has been written in the foregoing chapters, namely, the bronchologist through investigation of a bronchial lesion also may study the disease of the lung. Naturally, this does not mean that the bronchologist is able to evaluate in every case the type of pulmonary disease, but it does mean that if he observes a specific lesion in a bronchus, from this he may evaluate the character, severity, location, improvement, or aggravation of the disease of the pulmonary parenchyma.

Innumerable writers collected *indications* for bronchoscopy in cases of pulmonary tuberculosis. From these, two sets of indications have been selected here in which minor alterations have been made. The first collection of indications<sup>20</sup> informs the referring physician on how to decide in which cases he should request the help of a bronchologist. According to these principles, bronchoscopy is indicated in the following instances:

- 1 Recognizable bronchial stenosis
- 2 Bilateral changes, for determination of the side of the active process
- 3 Shrinking condensations
- 4 So called "healthy bacilli expectorators" for determination of the location of the lesion
- 5 Hemoptysis
- 6 Prior to pneumothorax if there is the slightest suspicion of tuberculous pneumonia or bronchial stenosis
- 7 Prior to every permanent collapse therapy

In the second collection of indications the referring physician presents his problem and specifies the aim of the bronchoscopy. These indications<sup>21</sup> are more useful to the bronchologist. It has been previously stressed that the bronchologist must know the problem of the referring physician; this collection shows indications from this point of view.

- 1 Suspicion of bronchial rupture of tuberculous intrathoracic lymph nodes
- 2 Suspicion of tuberculous bronchitis
- 3 Suspicion of bronchial stenosis
- 4 Hemoptysis
- 5 Obstruction therapy
- 6 Aspiration therapy
- 7 Examination of secretion
- 8 Examination prior to pulmonary surgery
- 9 Other questions

The questions of bronchoscopy in pulmonary tuberculosis will be discussed in this order.

*Contraindications* to bronchoscopy in pulmonary tuberculosis are a severely debilitated patient extensive laryngeal tuberculosis miliary tuberculosis and severe hemoptysis (Chaps 4 and 10)

### *Bronchial Rupture of Tuberculous Intrathoracic Lymph Nodes*

Older handbooks and papers on pathology considered rupture of a lymph node into a bronchus to be rare and in almost every case lethal. Until the middle 1930s writers overlooked the fact that the intruding material may cause atelectasis or pneumonia the patient eventually expectorated the granulation or caseous debris or at autopsy the secondary disease (pneumonia abscess bronchiectasis) dominated the picture and inspection of the bronchi was neglected.

Around 1919 the first mention appeared of particular extensive shadows due to pulmonary tuberculosis which suddenly arose in a pulmonary unit and then with the same rapidity disappeared. Only after 1930 was it realized that these shadows represented atelectases which were caused by compression or rupture of the contents of enlarged tuberculous lymph nodes.

By 1930 pathologic x ray and bronchoscopic investigations revealed that the rupture of lymph nodes into the bronchial lumens occurs more frequently than was formerly believed. Primary tuberculosis in most countries and in the majority of cases appears in infancy and childhood therefore rupture of tuberculous lymph nodes may be observed chiefly at these age levels. It may infrequently appear also in the adult if the patient acquires pulmonary tuberculosis only in his adulthood. The extremely rare primary tuberculous bronchitis (if it exists at all) may be mistaken for this lesion.

Generally lymph nodes of the lungs are situated on the outer bronchial wall. A caseous lymph node first grows upon the wall then destroys the cartilaginous layer protrudes into the mucosa narrows or obstructs the bronchial lumen and lastly intrudes through the mucosa and empties its content or a part of it into the bronchus. According to the contents granu

Rupture of caseous lymph nodes observed bronchoscopically follows the first symptoms (*mitis fever erythema nodosum*) of a primary pulmonary tuberculosis after 4 to 6 months in the average case. In infancy this period may be shorter because of weak resistance since tuberculosis at this age is similar to an acute contagious disease in its rapid onset. The actual or observed rupture of a tuberculous lymph node due to acquired pulmonary tuberculosis in an infant of 6 weeks (En, 68) Recently ruptures



FIG 64 A Bronchial rupture of a tuberculous lymph node caused atelectasis of the left lung of a four-year-old boy. By bronchoscopy a rubberlike cheese (C) 7 by 9 by 18 mm (C) was removed from the left main bronchus. B The situation 24 hours later.



FIG 65 A B Bronchial rupture of a tuberculous lymph node caused atelectasis in the right lung. C Removal of the rubberlike cheese. D E The situation 24 hours later.

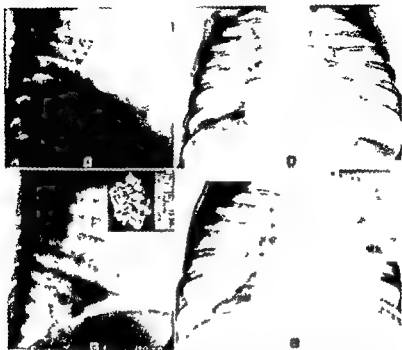


FIG. 68 A B Bronchial rupture of tuberculous lymph nodes first caused atelectasis in the right lower lobe then valvular emphysema in the right upper and middle lobes and also pneumothorax (C D) in a nine month-old infant. The atelectatic lower lobe is separated from the diaphragm by the pneumothorax (C). Note the valvular emphysema, atelectasis and pneumothorax with the displacement of the mediastinum and heart in one film (D). The upset status bronchoscopically removed bloody debris.

were observed simultaneously with erythema nodosum which raised a question as to whether these nodules really belong to the first stage of the disease<sup>24</sup>

**Signs and Symptoms** The symptoms of rupture of a lymph node are

- 1 Paroxysmal sonorous or hoarse cough
- 2 In case of stenosis of the trachea or a major bronchus the patient may be dyspnoic
- 3 Simultaneously with the rupture the respiration is stridorous
- 4 Changes in temperature in the blood picture in the sedimentation rate may be observed only in cases of purulent infection (pneumonia) or tuberculous dispersion to the pulmonary parenchyma
- 5 By x-ray examinations simple stenosis (Fig 11) valvular emphysema (Figs 13-17 66 and 68) and atelectasis involving a segment lobe or lung (Figs 64-67 and 69-76) may be observed. Naturally in cases of





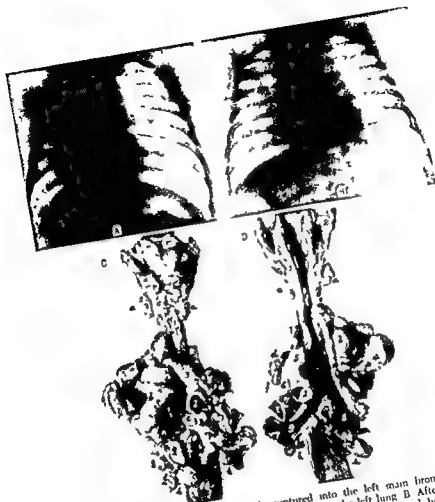
FIG. 67 Calcified tuberculous lymph node ruptured into the right middle lobe bronchus of a twenty-eight year old man and caused bronchiectasis in the middle lobe (B and E) A The calcified content of the lymph node (see arrow) C After bronchoscopy removal of the content only fragments (D) remained (see arrow)

bronchial rupture x ray signs may be absent it depends on the degree and location of the stenosis

Generally rupture of a tuberculous lymph node serves as a healing process by the elimination of tuberculous caseous material from the organism At the time of the rupture the general condition of a child usually is good often the child appears completely healthy

Upon observing the above symptoms after excluding the presence of a tuberculous infection (by negative reaction to tuberculin test) a foreign body must be considered first of all

**Therapy** Improved bronchoscopic technique extended routine bronchos



68 A Tuberculous lymph node ruptured into the left main bronchus of a 4-year-old boy and caused valvular emphysema in the left lung B After bronchoscopic removal of the cheesy debris the valvular emphysema disappeared but bronchophagical fistula arose because the same lymph node communicating with the left main bronchus ruptured also into the esophagus The infant died of bronchopneumonia Specimens of the larynx trachea and mediastinal organs C Anterior view D Posterior 1 Tracheostoma 2 aorta 3 innominate artery 4 common carotid artery 5 subclavian artery left 6 Botallus duct 7 left pulmonary artery 8 right main bronchus 9 left main bronchus 10 esophagus a paratracheal lymph node b tracheal lymph node c pretracheal lymph node d lymph node of the aorta e epibronchial lymph node f lymph node of Botallus duct g bifurcation lymph node (cut) h bronchopulmonary lymph node i cavity of the bifurcation lymph node Threads are introduced into the perforations of the main bronchus and esophagus

copy to infants and children with primary tuberculosis. Today bronchoscopy is applied in these cases as a harmless procedure for diagnostic and therapeutic purposes from earliest infancy.

In primary tuberculosis, proved stenosis indicates bronchoscopy. These indications are simple stenosis with Holzknecht's sign, eventually with stridor or dyspnea, severe dyspnea itself, valvular emphysema (Figs 13-17, 66, and 68), and atelectasis (Figs 64-67 and 69-76). Bronchoscopy should be performed in cases of excited, sonorous, bitonal cough when rupture in the tussigenic zone near the carina tracheae is suspected. Sudden onset of a status asthmaticus in a tuberculous child also indicates bronchoscopy. It may be due to bronchial rupture. Rupture must be particularly considered if the asthmatic state is not influenced by the usual medication.

The primary tuberculous changes may leave behind bronchiectasis, lymph node cavities, or specific bronchitis at the place of perforation. These are the so-called 'postprimary' changes, which seem to be independent of the pulmonary process. In these cases the patient keeps coughing slightly, x rays are negative, but sputum is positive. These patients are the so-called "healthy bacilli expectorators." Bronchoscopy may reveal the source of the discharge of bacilli. However, if it does not, bacteriologic examination of secretion aspirated separately from each lung may lead closer to the lesion. For example, after this information is obtained thin walled hidden cavities may be demonstrated on anteroposterior or lateral planigrams taken in the proper projection.

In some selected cases caseous lymph nodes may be removed by external surgery.



FIG. 69. Chest x rays of a seven year-old girl showing epituberculosis in the anterior segment of the right lung due to bronchial rupture into the anterior segmental bronchus. A Lateral B Anteroposterior. Note the distinct and sharp border in the interlobar (horizontal) fissure and indistinct border in the intersegmental septum.



FIG 70 X-rays of a one and a half year old boy showing epithelioid tuberculosis in the hilar segment of the right upper lobe due to bronchial rupture of a tuberculous lymph node. A Lateral B Anteroposterior

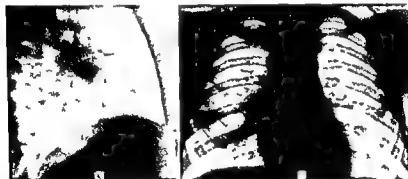


FIG 71 X-rays of a four year old girl showing a shrunken lateral segment of the right middle lobe due to fibrosis caused by epithelioid tuberculosis. A Lateral B Anteroposterior. Note the distinct and sharp border in the interlobar fissures and indistinct border in the intersegmental septum.

### *Tuberculous Bronchitis*

The term *tuberculous bronchitis* refers to specific lesions of the bronchial mucosa and the underlying bronchial wall.

Tuberculous disease of the bronchus was known even before the discovery of the tubercle bacillus but its importance has been recognized only in the last two decades.

Tuberculous disease of the pulmonary parenchyma in almost every case extends to minor bronchi of the affected area and to the nearest draining bronchus. The bronchologist observes only lesions of the bronchoscopically



FIG 72 X ray films showing the shadow of the medial segment of the right middle lobe of a four year old boy due to epituberculosis A Lateral B Anteroposterior



FIG 73 X rays of a six year old boy showing epituberculosis in the apical segment of the right lower lobe due to bronchial rupture of a tuberculous lymph node A Lateral B Anteroposterior



FIG 74  
lung of  
veloped

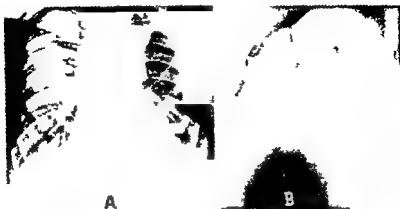


FIG. 75 X-ray film and planogram show massive shadow of the truncated lobe of the left lung of a thirteen-year-old boy due to apical tuberculosis. A Anteroposterior. B Lateral.

visible bronchi; thus, he finds tuberculous bronchitis only in a certain percentage of patients suffering from pulmonary tuberculosis. Its incidence has been variously stated as between 7 per cent and 26 per cent. The rate is actually more likely to be closer to the lower figure and roughly 10 per cent is an acceptable figure.

Three types of tuberculous bronchitis are found: exudate, caseous, ulcerative, and productive. Variations of all three are known.

Interchymal infection extends to the bronchial wall from the parenchyma, mostly through the draining bronchus, upon the surface from lymph nodes through the bronchial wall, and by transportation through lymph vessels under the mucosa. Hematogenous tuberculous bronchitis is very rare.

Inflammation of the bronchial mucosa frequently is nonspecific. This inflammation is often difficult to differentiate from the tuberculous type.

The most silent symptoms of tuberculosis of the trachea and major bronchi are excited, spasmodic cough in asthmatic state or alternately severe dyspnea, crepitation and stridor audible near the patient, and sputum sometimes blood streaked (Fig. 77).

Every type of tuberculous bronchitis is curable with the usual medication for tuberculosis, such as streptomycin, para-aminosalicylic acid (PAS) and isoniazid (INH). Local applications of silver nitrate and lactic acid solutions and electrocautery are out-of-date procedures. Blanching of the area with epinephrine is sometimes advantageous. Large masses of granulation tissue should be removed and the area treated with electrocautery. Removed tissues should be examined histologically in every case. Such an examination is very important because a tuberculous patient may



Fig. 76 X rays showing atelectasis in the lingula due to compression of the lingular bronchus caused by tuberculous lymph nodes. A Anteroposterior B Lateral Note the lymph nodes which compressed the lingular bronchus in the plain film (C)

may have a bronchial carcinoma. Conversely a lesion appearing grossly to be carcinoma may prove histologically to be tuberculosis. The biopsy is generally harmless, only in rare cases has bronchogenic dissemination occurred. Such minor hazards in questionable cases must be accepted.

#### *Tuberculous Bronchostenosis*

In pulmonary tuberculosis cicatricial stenosis appears after healing of specific bronchitis. The tuberculous inflammation causes destruction and later cicatrization and shrinkage in the bronchial wall, particularly in the mucosa and fibrocartilaginous layer (Fig. 78). The cicatrization gen-

erally affects the mucosa, but usually it looks red, congested, and smooth without any scar tissue due to greater destruction in the deeper layers. In some rare cases, rupture of lymph nodes also causes stenosis.

Cicatricial stenosis has been found in from 2 to 19 per cent of tuberculous patients.

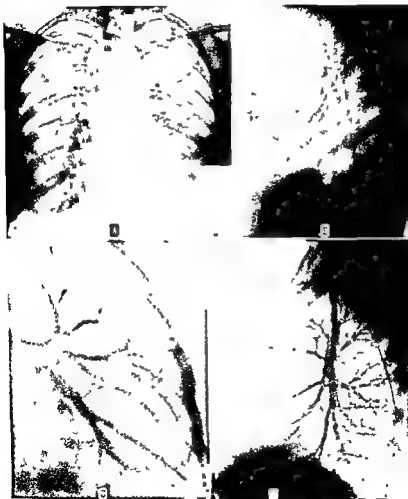


FIG. 7. Chest x-rays failed to reveal any lesion in the lungs of a fourteen-year-old girl who expectorated blood-streaked sputum for months. A: Anteroposterior. B: Lateral. C, D: Bronchograms showing obstruction of the lateral and posterior basal segmental bronchi of the left lower lobe. Bronchoscopic biopsy proved tuberculous bronchitis. The lesion was treated with antibiotics and cured.





FIG. 76 X rays showing atelectasis in the lingula due to compression of the lingular bronchus caused by tuberculous lymph nodes. A Anteroposterior B Lateral. Note the lymph nodes which compressed the lingular bronchus in the plumbigram (C)

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#### ***Tuberculous Bronchostenosis***

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erally affects the mucosa but usually it looks red, congested and *smooth* without any scar tissue due to greater destruction in the deeper layers. In some rare cases rupture of lymph nodes also causes stenosis.

Cicatrical stenosis has been found in from 2 to 19 per cent of tuberculous patients.



FIG. 77. Chest x-rays failed to reveal any lesion in the lungs of a fourteen-year-old girl who expectorated blood-streaked sputum for months. A: Anteroposterior. B: Lateral. C: D: Bronchograms showing obstruction of the lateral and posterior basal segmental bronchi of the left lower lobe. Bronchoscopic biopsy proved tuberculous bronchitis. The lesion was treated with antibiotics and cured.



FIG. 76 X rays showing atelectasis in the lingula due to compression of the lingular bronchus caused by tuberculous lymph nodes. A Anteroposterior B Lateral. Note the lymph nodes which compressed the lingular bronchus in the planigram (C)

also have a bronchial carcinoma. Conversely, a lesion appearing grossly to be carcinoma may prove histologically to be tuberculosis. The biopsy is generally harmless, only in rare cases has bronchogenic dissemination occurred. Such minor hazards in questionable cases must be accepted.

### *Tuberculous Bronchostenosis*

In pulmonary tuberculosis cicatricial stenosis appears after healing of

ectasis benign or malignant tumor abscess etc (Figs 56 77 and 85) In tuberculous patients hemoptysis is chiefly caused by dissolving tissues e.g. by a fresh tuberculous cavity. Ulcers or granulations of the bronchial mucosa seldom bleed. Occasionally perforation of a tuberculous lymph node causes hemoptysis.

In hemoptysis the pneumologist internist general practitioner and bronchologist are interested in the source of bleeding. Blood discharged from a bronchus leads to the source.

Hemoptysis does not contraindicate bronchoscopy. On the contrary the proper time to make the examination is simultaneously with the hemoptysis (Chap. 4).

Weeks or months after bleeding has stopped the examination is not so informative. In cases of severe bleeding it is better to postpone bronchoscopy because the patient generally is in a state of shock, anesthesia is inadequate and inspection is impaired (see Contraindications in Chap. 4). However even in these cases one should not wait until the bleeding completely stops. If the patient's general condition and diminution of the bleeding allow bronchoscopy should be performed and the source of the bleeding determined without delay. If the examination in a bloodless interval has negative results it is best to perform another bronchoscopic examination when hemoptysis recurs. To make another controversial statement it also may happen that very dangerous bleeding can be stopped only by emergency bronchoscopic tamponade.

Blood clots may occlude the lumen of a major bronchus causing atelectasis. It is urgent that these clots be removed.

Search for the source and the cause of hemoptysis is the duty of the bronchologist. It must be continued by all the means at his disposal until success is achieved. No proper therapy can exist without a determination of the source and cause. It must not be forgotten that in a tuberculous patient hemoptysis may be due to another disease e.g. foreign body bronchiectasis or tumor. An example of the searching method of a bronchologist is given in Chapter 8 Case 7. However it can happen that the most ambitious search will end unsuccessfully.

### ***Obstruction Therapy***

Numerous investigators have observed that a tuberculous cavity may heal after obstruction of the draining bronchus. The draining bronchus is frequently affected by tuberculous inflammation. After its recovery cicatrization may obstruct its lumen, the wall of the cavity becomes thin, air is absorbed and the cavity will slowly be transformed into cicatricial tissue. Sometimes as granulation tissue progresses toward scar formation some calcification arises in the wall of the cavity. This healing process may be favored by degeneration of the aerobic mycobacteria trapped in the



FIG 78 Bronchograms showing cicatricial stenosis of the left main bronchus and occlusion of the left upper lobe bronchus due to antecedent bronchilitis cured by antibiotics. A Anteroposterior B Lateral

Symptoms of bronchial stenosis are stridor simple stenosis with Holz knecht's sign valvular emphysema or atelectasis (Chap 5) Sooner or later bronchiectasis arises in the distal parenchyma (Fig 79)

By using dilatation therapy on the scarred stenosis the bronchologist only causes harm because of repeated injuries and consequently further cicatrization. The cicatricial bronchial wall is more rigid than the esophageal one and thus is less suitable for dilatation therapy. Reasonable treatment of a cicatricial stenosis is resection of the stenosed portion of the bronchus and anastomosis of the involved bronchi. By this method distal parenchyma can be saved if it is intact. If it is not the distal portion has to be resected. The bronchologist may diminish distal suppuration by aspiration of the stagnating pus but in only a few cases is he able to stop exudation. Retained secretion is aspirated not only during every diagnostic bronchoscopy but also for therapeutic reasons. The improvement achieved by this treatment is in general only temporary. Aspiration and diminution of the pus is important chiefly in preparing the patient for surgery.

### *Hemoptysis*

It was mentioned earlier that in the majority of cases hemoptysis is not due to pulmonary tuberculosis. It originates more often from bronchi

closed cavity. In rare cases anaerobic mixed organisms may impair healing of the cavity.

On the basis of these experiences, bronchoscopic obstruction of the draining bronchus was attempted as a means of healing the tuberculous cavity. Experiments were done with electrocauterization and application of 50 per cent silver nitrate solution, but a grossly healthy bronchus could very seldom be obstructed or narrowed to such a degree that the tuberculous cavity recovered. Obstruction therapy is still in the state of experimentation. The author does not accept patients for this therapy but it is mentioned here because of its frequent appearance in the medical literature.

### *Aspiration Therapy*

Tuberculous cavities that do not collapse and recover with the usual therapy (including pneumothorax and pneumoperitoneum) are well known. Two evident causes of this phenomenon exist: valvular inflation and cicatricial rigid wall. In cases of rigid wall there is little hope of benefit by aspiration therapy but the inflated cavities may be cured.

In therapy of the pulmonary abscess aspiration treatment is a conventional procedure (Chap. 8). This pattern has been applied in the therapy of tuberculous pulmonary cavities. The aim is to abolish the valvular action. This can occur with simple aspiration of the secretion. This secretion alone is able to keep valvular action in existence. Aspiration therapy may also eliminate the mucosal inflammation causing valvular obstruction.

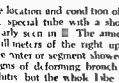
The bronchologist must be familiar with this treatment because pneumologists frequently call for it. Nevertheless the expected result will be obtained only in some fortunate cases.

### *Examination of Secretion*

Bronchoscopically sterile aspiration of the bronchial secretion and its bacteriologic examination are very important procedures. Secretion must always be obtained from the affected portion of the lung. Thus specimen which is not diluted by secretions coming from other bronchi contains mycobacteria in the greatest attainable concentration.

Through a bronchoscope secretion from the two lungs may be aspirated separately. This method can determine which side has the active process.

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the location and condition of the right upper lobe (D and F). The bronchoscope and the special tube with a short right angle curve used for aimed bronchography are clearly seen in . The aimed bronchograms (D and E) revealed that the first few millimeters of the right upper lobe bronchus were extremely narrowed; bronchi of the anterior segment showed moderate bronchiectasis; the remaining bronchi showed signs of deforming bronchitis. Bronchiectasis was developing from deforming bronchitis but the whole lobe was still aerated. Leftectomy was performed.



A



B



C



D



E

FIG. 79 Tuberculous bronchitis caused severe mucosal stenosis about 1 mm in diameter of the right upper lobe bronchus which was diagnosed and observed bronchoscopically for 2 years. The patient refused surgical treatment at that time. From the fairly old stenosis and the large amount of thick mucopurulent secretions.

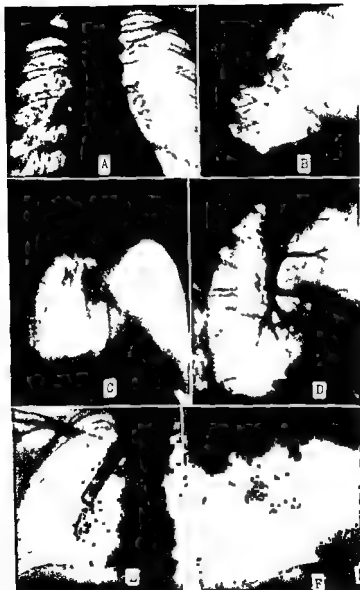


FIG. 80 A tuberculous cavity in the apical segment of the right lower lobe of a thirty nine year-old woman was cured with antibiotics but after about 6 months cough and low grade fever returned. Chest x rays (A and B) and plainograms (C) failed to demonstrate cavity in the apical segment. D Normal bronchogram showing dead tree effect in this segment. E & F With aimed bronchography a walnut sized cavity filled in the apical segment. Segmental resection was performed.



Naturally these examinations do not produce absolutely accurate results. Secretions from the affected side may flow into the other side. The discharge of bacilli is not continual; therefore a negative finding is not valid as evidence. However, these examinations usually reveal the real situation with a great degree of certainty.

### *Examination Prior to Surgery*

It is an imperative rule for the thoracic surgeon to call for bronchoscopy before every operation. For example, in cicatricial stenosis he is obliged to perform resection of the distal parenchyma instead of thoracoplasty. In tuberculous bronchitis it is advisable to cure the bronchial disease prior to operation because of the danger of the development of a bronchial fistula.

### *Other Questions*

The pneumologist and the general physician may ask other questions of the bronchologist. For example, they may ask whether there is a tuberculous bronchitis or cicatricial stenosis in the draining bronchus of the cavity, indicating pneumothorax therapy, or whether a tuberculous bronchitis perpetuates a bronchial fistula. They may call for bronchoscopy if the tuberculous origin of a given pulmonary disease is questionable, or may request aimed bronchography to determine the presence of a tuberculous cavity (Fig. 60). The pneumologist may call for a comparative examination of his therapeutic result, and so on. In the foregoing paragraphs only the most frequent requests have been discussed.

### *Complications*

In a tuberculous patient bronchoscopy causes slight elevation in temperature in about 10 per cent of the cases. With the patient in the hands of a skilled bronchologist with good manual ability, high fever or complications demonstrable by x-ray examination may occur, but usually they do not in more than 0.5 per cent of the cases. These complications are observed chiefly after biopsy and in cases of cicatricial stenosis and tuberculous bronchitis. The rare complications closely following bronchoscopy are in all probability caused by congestion of the injured mucosa and consequent stagnation of secretion in the distal parenchyma. From these complications smaller pneumonic foci may arise.

## MYCOSIS

Recently interest in mycosis has increased. There are two types of mycotic infection: a primary or exogenous infection entering from out-

million or more units daily) or Aureomycin hydrochloride Terramycin chloramphenicol INH in cases of actinomycosis iodine therapy with desensitization in cases of aspergillosis and withdrawal of antibiotics and steroids with administration of potassium iodide in cases of mucormycosis

### Summary

In tuberculous patients bronchoscopy may be indicated where there is suspicion of bronchial rupture of tuberculosis lymph nodes suspicion of tuberculosis bronchitis suspicion of bronchial stenosis in cases of hemoptysis for obstruction and aspiration therapy for examination of secretion prior to surgery and in other conditions

Generally bronchial rupture of a tuberculous lymph node serves as a healing process by elimination of caseous material and appears with relative frequency in infants and young children A caseous lymph node first grows upon the outer bronchial wall then destroys the cartilaginous layer protrudes into the mucosa narrows or obstructs the bronchial lumen and lastly empties its content into the bronchus

Improved bronchoscopic technique has extended routine bronchoscopy to infants and young children In primary tuberculosis every proved stenosis indicates bronchoscopy Bronchoscopy should be also performed in cases of excited sonorous bilateral cough and sudden onset of a status asthmaticus

Bronchoscopy reveals tuberculous bronchitis in about 10 per cent of patients with pulmonary tuberculosis This disease is curable with the usual medication such as administration of streptomycin PAS or INH Local application of silver nitrate or lactic acid and cauterization are out of date procedures In cases of granulation tissue removal and histologic examination are very important

Cicatricial bronchial stenosis has been found in 2 to 19 per cent of tuberculous patients Bronchoscopic dilatation causes more harm than benefit Surgery is the reasonable treatment

work is the duty of a bronchologist

Numerous investigators have observed that a tuberculosis cavity may heal after the obstruction of the draining bronchus Bronchoscopic obstruction may be attempted this therapeutic procedure however is still in the state of experimentation

Bronchoscopic aspiration treatment of an inflated tuberculous pulmonary cavity may be successful The aim of this procedure is to abolish the bronchial valvular action In some fortunate cases this can occur with simple aspiration of the secretion

side, and a secondary, or endogenous, infection caused by common saprophytes in the patient

Pulmonary infections entering from the outside most frequently are due to *Histoplasma capsulatum*, *Blastomyces dermatitidis*, *Coccidioides immitis*, and *Nocardia asteroides*

The antibiotics by destruction of the intestinal bacteria interfere with synthesis of B vitamins and create favorable conditions for the proliferation of otherwise harmless fungi. These fungi attack, particularly, patients debilitated by other diseases (carcinoma, tuberculosis, diabetes, etc.) The most frequently found pathogenic endogenous fungi are *Aspergillus fumigatus*, *Actinomyces bovis*, and *Rhizopus* (pathogenic in mucormycosis)

**Signs and Symptoms.** In acute pulmonary histoplasmosis symptoms resemble primary atypical pneumonia. Roentgenograms show often diffuse mottled densities uniformly scattered throughout both lung fields. Enlarged hilar lymph nodes are typical.

In blastomycosis, many variations of symptoms may be observed (productive cough, loss of weight, moderate fever and leukocytosis, high sedimentation rate, chest pain). Bronchitis, tracheitis, and miliary pulmonary involvement may also appear.

In coccidioidomycosis the primary pulmonary lesion is most commonly asymptomatic. In these cases the only diagnostic value is the skin test. Frequent symptoms are similar to those in influenza (cough, fever, pain in chest, malaise, rarely hemoptysis). Lesions seen on roentgenograms are bronchopneumonic densities, thin-walled cavities, hilar lymphadenopathy, or small dense nodules.

In nocardiosis the symptoms and physical and roentgen signs resemble pulmonary suppuration.

In aspergillosis, the usual symptoms are those of acute tracheobronchitis. In toxic forms, cough, fever, prostration, may be found. In organizing pneumonia symptoms resemble tuberculosis.

In mucormycosis, pneumonic inflammation, infarction, and intralobar invasion of the bronchial wall with penetration into the hilar tissues, pulmonary artery, and vein may be found.

Exogenous fungi demonstrated in sputum verify the diagnosis. Their presence in the oral cavity is considered pathogenic. Fungi of the endogenous group must be proved to be in secretion obtained bronchoscopically by sterile aspiration. Their presence in the sputum alone is not valid as evidence of their pathogenicity.

**Therapy.** Symptomatic therapy alone may be used in cases of histoplasmosis and coccidioidomycosis, saturated potassium iodide, stilbimi-

# Bronchial Tumors

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Almost every pulmonary tumor arises in a bronchus. Perhaps only the alveolar cell carcinoma originates in the pulmonary parenchyma. Bronchial tumors are divided into three groups: *benign tumors* such as granuloma, neurofibroma, lipoma, papilloma, osteoma, hamartoma, chondroma, angioma; *borderline tumors* such as adenomas of the solid type (carcinoid) and glandular type (cylindroma); and *malignant tumors* such as carcinoma and sarcoma.

## *Benign Tumors*

Essentially benign tumors are relatively rare. Among such tumors only chondromas have a known tendency toward malignant degeneration. An endobronchial benign tumor can be removed bronchoscopically even if it is attached by a broad base. With bronchoscopic scissors, knife, or snare it can be separated from the bronchial wall. Its removal is generally not difficult (Figs 81-84). However, angiomas located in the bronchial wall indicate external surgery (Fig 85).

## *Borderline Tumors*

The borderline tumors (the two types of adenoma) deserve a special place among the bronchial tumors. The adenoma is epithelial in origin. The cells are cuboidal and generally uniform with rare mitotic figures. This very slowly growing tumor never becomes necrotic in its center as a malignant tumor does, even if its extent is extremely large.

Two forms are differentiated: the solid type or *carcinoid* in which type the cells entirely fill the space in the stroma (Fig 86A) and the glandular type or *cylindroma* in which the cells form one, two, or more layers in glandular pattern (Fig 86B). The cylindroma is considered by many writers to be less benign than the carcinoid type.

Before 1880 this tumor was found only accidentally at autopsies and because of its character as seen on histologic examination it was classified as a carcinoma or an adenoma that had undergone malignant degeneration and had in fact become a carcinoma. Between 1882 and

Sterile bronchoscopic aspiration of the bronchial secretion and its bacteriologic examination are very important procedures. In cases of pulmonary tuberculosis this method may succeed in detecting which side the active process is on.

It is imperative to call for bronchoscopy before every lung operation.

Complications after bronchoscopy of a tuberculous patient are observed in about 0.5 per cent of the cases chiefly in connection with biopsy, cicatricial stenosis and specific bronchitis.

There are two types of mycotic pulmonary infections: primary or exogenous and secondary or endogenous. Exogenous infections are most often due to *Histoplasma capsulatum*, *Blastomyces dermatitidis*, *Coccidioides immitis* and *Nocardia asteroides*. Fungi of the endogenous group must be proved to be in the bronchial secretion obtained bronchoscopically by sterile aspiration.



FIG 82 X rays showing some irregular densities in the right upper lobe region of a forty five-year-old man who was for several months moderately dyspnoic A Anteroposterior B Lateral C Bronchoscopy and bronchography showed a roundish smooth tumor in the right main bronchus the upper lobe bronchus was obstructed D The oval tumor 14 by 16 by 29 mm was removed bronchoscopically histologic examination showed fibrolipoma E No bronchiectasis developed in the distal parenchyma



FIG 81 X rays showing atelectasis of the right upper lobe of a twenty month-old boy A Lateral B Anteroposterior This lesion was considered to be ep tuberculous and was treated with streptomycin and INH Reaction to tuberculin test with 1:100,000 solution was positive C By bronchoscopy a pedunculated tumor 3 by 6 by 9 mm was removed from the upper lobe bronchus where it had partially protruded into the main bronchus The sinking upper lobe expanded within two months Diagnosis based on histologic examination of the tumor was nonspecific granuloma

1930 pathologists recognized its benignity and separated it from the carcinomas After 1930 this tumor was known to be benign it was also recognized endoscopically and removed through the bronchoscope After 1940 searchers detected that the adenoma frequently penetrated through the bronchial wall Therefore surgeons too are concerned with its treatment tumors are removed by external operation as well as through the bronchoscope

The question of the benignity or malignancy of the adenomas is still unsettled Some writers consider them definitely benign some acknowledge that during its long period of growth the adenoma may undergo malignant degeneration others believe it is actually or potentially malignant (Fig 87)

It is generally believed that the adenomas are derived from the epithelium of acini or efferent ducts of the bronchial glands or basal cells of the bronchial mucosa If the tumor originates from glands lying inside the cartilages it grows endobronchially (Figs 87-89) if the involved glands are located in the wall among the cartilages the tumor grows intramurally (Fig 92B) and lastly if the adenoma starts to grow outside the cartilages where glands also exist (Chap 1) only its smallest portion emerges into the bronchial lumen above the surface and the larger portion extends into the pulmonary parenchyma This latter is called the "iceberg type" of adenoma (Figs 90 and 91)

Bronchial adenomas occur in 5 per cent of all cases of bronchial tumor

**INITIAL PERIOD** In the very beginning symptoms are absent. Later they are related to the endobronchial growth of the tumor. The irritating effect of the tumor may cause coughing attacks which are sometimes asthmatic. Adenomas often bleed from a network of blood vessels located on the surface. This bleeding differs from the carcinomatous hemoptysis. In carcinoma the sputum is blood streaked or like raspberry jam (micro hemoptysis), the adenoma type is repeated profuse, and sometimes perilous. In females hemoptysis often appears at the time of the menses or in the premenstrual period. The stenosed lumen may result in exertional dyspnea and stridor, but in this period the condition of the patient is still good, and the blood picture and sedimentation rate are normal.

**PERIOD OF SEVERE BRONCHIOSTENOSIS OR TEMPORARY OCCLUSION** In this period valvular emphysemas, recurrent atelectasis, pneumonias or pleuro pneumonias are observed. Bronchiectasis may develop in this period also.

**PERIOD OF PERMANENT OCCLUSION WITH SUPPURATIVE COMPLICATIONS** In this period bronchiectasis, abscesses, gangrene, and empyemas are observed.

Some authors\*\* speak directly of the "adenoma syndrome." This syndrome is represented by simultaneous bronchostenosis and hemoptysis in young individuals. Long duration of the symptoms is very characteristic. It may last from 30 to 40 years. Therefore adenoma must be considered in every case of prolonged pulmonary suppuration.

**Diagnosis** X-ray symptoms of adenoma are not characteristic. They are similar to that of other types of bronchial stenosis. These symptoms are given in detail in the discussion of pathological respiratory mechanisms in Chapter 3. The shadow of the growth is pictured on plinigrams and bronchograms, sometimes penetration of the tumor into the pulmonary



FIG. 84 A Anteroposterior x ray showing a round tumor in the right chest cavity at the diaphragmatic level. There was no sign of any lesion in the left chest cavity. The tumor with a somewhat irregular wall was removed bronchoscopically and on the right side was treated by



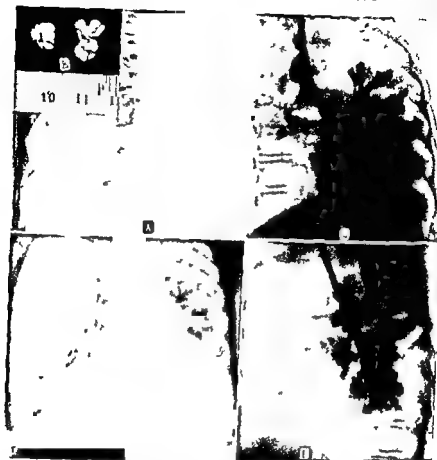


FIG. 83 A Anteroposterior x ray showing atelectasis in the left lung of an eight year old girl who was treated for years for laryngeal papilloma. Papilloma with the same structure obstructed the left main bronchus. E This was removed bronchoscopically. X ray (B) and bronchograms (C and D) taken 11 days after bronchoscopic removal of the tumor demonstrate saccular bronchiectasis in the left lung. Pneumonectomy was performed.

and 75 per cent of benign tumors are adenomas. Any general practitioner may encounter this type of tumor.

They occur at every age having been observed in three year-old children as well as in patients over sixty. However they occur more frequently between the ages of twenty and thirty. The most common location is in the major bronchi (main and lobar bronchi) and in the trachea they appear less frequently in a segmental bronchus or in the periphery of the lung.

Three periods are differentiated in the clinical course of the bronchial adenoma.

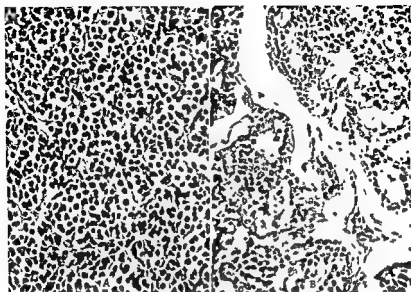


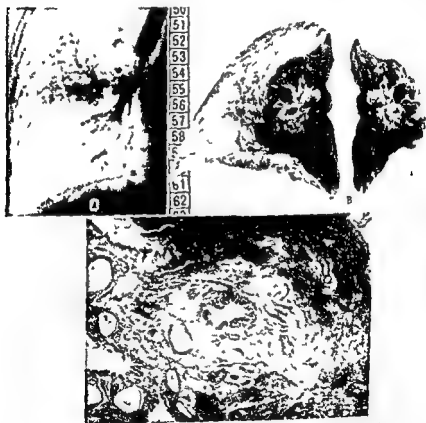
FIG. 86 Microphotographs of (A) a solid or circumscribed type of bronchial adenoma and (B) a glandular or cylindroma type.



FIG. 87 Right pneumonectomy was performed in a case of bronchial adenoma in a thirty-seven year old man. A The pedunculated tumor of the stem bronchus (see arrow to) *brim*, stem bronchus *brlm*, middle lobe bronchus and *brlt*, lower lobe bronchus. B The tumor attached to the bronchial wall by a narrow base. The tumor indicated bronchoscopic surgery. But in spite of the protest of the bronchologist the surgeon performed pneumonectomy because adenoma has been considered by prominent writers to be actually or potentially malignant. This misunderstanding of the intention of these writers resulted in unnecessary disability due to loss of a whole lung.

parenchyma may also be demonstrated on planigrams (Figs 90 and 93). However, none of these proves the diagnosis of adenoma.

*Bronchoscopy* is the most essential diagnostic procedure. Actually only bronchoscopy renders clinical recognition of an adenoma possible. The bronchologist in the majority of cases recognizes the adenoma from the bronchoscopic appearance alone. Its general appearance is polypoid, resembling a roundish tumor with scattered branching superficial vessels. Its surface is usually smooth, sometimes slightly uneven, bumpy, and in



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subsegmental bronchus was seen to be stenosed and with adenoma was diagnosed

histologically to differ within the same tumor. Therefore, a large specimen or, if possible the whole endobronchial tumor must be removed for histologic examination. Incorrect diagnosis of carcinoma is primarily due to the fact that the histologic findings do not coincide with the clinical picture or for other reasons are uncertain the biopsy must be repeated, perhaps many times. As was mentioned among the symptoms the adenoma may bleed profusely therefore preparation must be made for bronchoscopic hemostasis even if biopsy only is to be performed.

**Therapy** The following methods may be used for extirpation of a bronchial adenoma: endoscopic removal, bronchotomy, bronchial resection, anastomosis and plastic repair, and pulmonary resection (segmental resection, lobectomy, pneumonectomy). From these methods the one most suitable for a given case should be selected.

The following methods may be used for extirpation of a bronchial adenoma: endoscopic removal, bronchotomy, bronchial resection, anastomosis and plastic repair, and pulmonary resection (segmental resection, lobectomy, pneumonectomy). From these methods the one most suitable for a given case should be selected.

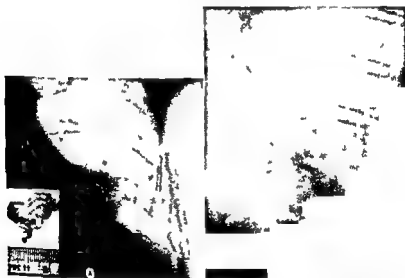


FIG. 89. X-ray films showed pneumonia in the posterior basal segment of the left lower lobe of a forty-six-year-old man. A: Anteroposterior. B: Lateral. Pneumonia recurred every month for 2 years (twenty-four times). The last physician requested bronchoscopy. C: A 10 by 12 by 22 mm tumor attached by an 8 mm pedicle to the bronchial wall was removed. This was diagnosed histologically as a solid (carcinoid) type of adenoma. Chest x-ray taken 2 weeks after removal of the tumor showed some densities in the posterior basal segment. This case represents a frightening example of neglect of indications for bronchoscopy since the frequently recurring segmental pneumonia should have called attention to the possibility of bronchial stenosis.

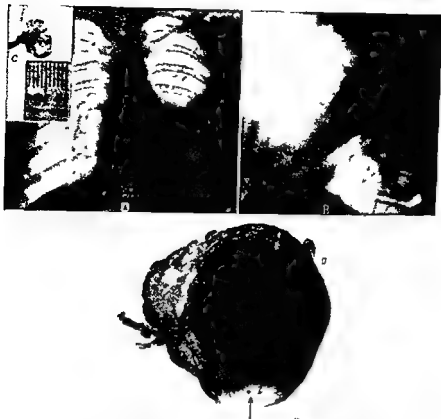


FIG 88 Chest x rays of a sixteen year-old girl showing atelectasis in the axillary subsegment of the left upper lobe A Anteroposterior B Lateral Bronchoscopy revealed a tumor in the right main bronchus and a small piece of tumor tissue was removed for histologic examination carcinoma was diagnosed Biopsy was repeated twice with the same result then a consultation confirmed the former diagnosis and pneumonectomy was advised The frightened mother brought the girl for bronchoscopic examination C D A 6 by 6 by 10 mm pedunculated tumor was removed and diagnosed as a bronchial adenoma by histologic examination D Note the thick capsule of the tumor composed of connective tissue the arrow points to a defect which could be the site of the previous biopsy The thick capsule and the small size of the removed biopsy specimen had resulted in the histologic error The coincidental atelectasis of the axillary subsegment later disappeared

rare instances imitates ■ blackberry The color, depending on the vascularization is whitish or yellowish pink very rarely red or even lilac

For accurate diagnosis the bronchoscopically removed specimen ■ essential Proper excision of the specimen ■ very important The capsule of the adenoma ■ often thick, fibrous or edematous the forceps must bite deeply into it for valuable material (Figs 88 and 90) The adenoma ■ scarcely recognizable from the few cells removed with the thick fibrous capsule (Fig 88) Moreover, the structure of an adenoma may be seen

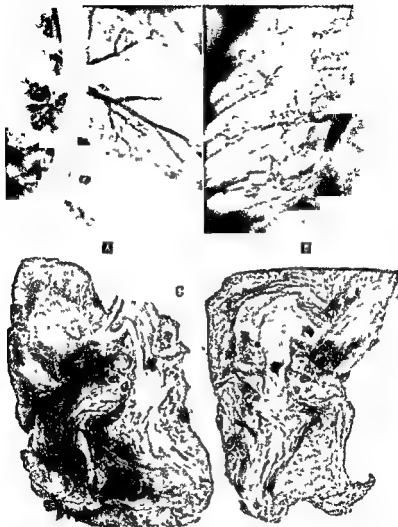


FIG. 91 A shrinking atelectatic process in the right middle and lower lobes was due to a hazelnut sized adenoma which was removed by bronchoscopic surgery from the right stem bronchus. Bronchoscopic reexamination and lateral (A) and anteroposterior (B) bronchograms showed bronchiectasis in the right middle and lower lobes. The caliber of the stem bronchus was normal. Right pneumonectomy was performed instead of lobectomy because the healthy upper lobe could not be isolated from the bronchiectatic middle and lower lobes. The surgical specimen (C) proved to be a tumor of the iceberg type and unsuitable for bronchoscopic surgery.

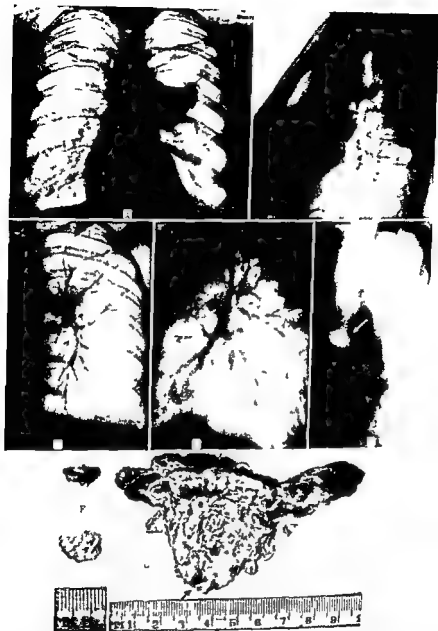


FIG 90 Chest x rays showing circumscribed shadow the size of a male fist in the left chest cavity in contact with the hilus and anterior mediastinum A Anteroposterior B Lateral Bronchoscopy revealed a smooth reddish tumor in the left main bronchus protruding from the upper lobe bronchus ample specimen of tissue removed to be the solid (carcinoid) type demonstrate obstruction C The removed upper tumor tissue

A case of bronchial adenoma unsuitable for endoscopic operation and cured with bronchial resection anastomosis and simultaneous lobectomy is demonstrated in Figure 93. Another case in which the indication for external surgery was too radical is shown in Figure 87.

The bronchologist in external operations assists the thoracic surgeon in many ways. By localization of the adenoma and determination of its relation to the neighboring tissues he is helpful in the selection of a suitable surgical method. He restores drainage of the distal lung by removal of the endobronchial portion of an iceberg type of adenoma; he diminishes or often stops exudation. In all these ways he helps the surgeon greatly. During the actual operation aspiration of blood and secretion is often necessary. Lastly the bronchologist assists in the postoperative care in all cases noted in the foregoing paragraphs; he may frequently be called upon for postsurgery bronchial aspiration.

### *Malignant Bronchial Tumors*

The incidence of pulmonary carcinoma has increased greatly in the last two decades. The majority of authors concede that this increase is truly marked and that the proved increase is due not just to earlier recognition of the tumor or to the improvement of diagnostic means. Until the end of the last century excellent pathologists performed autopsies in many thousands of cases and found pulmonary carcinoma in only from 0.05 to 0.06 per cent; this figure has risen now to 1 per cent of all autopsied cases or a percentage twenty times greater than some decades ago. Pulmonary carcinoma occurs now with a frequency at least equal to that of stomach malignancies and perhaps actually occurs more frequently.

According to their location pulmonary carcinomas are divided into four groups: *central* or *hilar* carcinoma—tumor of main and lobar bronchi; *intermediate* carcinoma—tumor of tertiary, quaternary or quintary bronchi; *peripheral* carcinoma—near the pleura; and *diffuse* carcinoma.

In gross appearance carcinoma presents a slight transparent yellowish white hard irregular mass. Its border is commonly not sharp, but some times on the cut surface it appears as though it were encapsulated.

Some decades ago the histologic architecture of the tumor was emphasized. On this basis a carcinoma was diagnosed to be a scirrhous carcinoma, carcinoma simplex, medullary carcinoma, adenocarcinoma, etc. In recent years physicians have not been satisfied with such a diagnosis because they are interested in the degree of malignancy of a tumor when considering the question from a clinical point of view. The degree of malignancy can be analyzed from its histologic structure, i.e. from the differentiation of the tumor cells. Today, according to this principle, two classifications are used. In the first system bronchial carcinomas are divided into three groups: *plano-cellular* (squamous cell) carcinoma,





FIG 92 Sections of the right stem bronchus showing failure of bronchoscopic removal of bronchial adenomas. A The entire endobronchial tumor was removed from a thirty-five-year-old woman; no pathologic condition was found by either bronchoscopic or histologic examination in the bronchus or inside the cartilage but in the peribronchum a large island of the tumor still existed (see arrows). B In a fifty-seven-year-old female normal lumen of the bronchus was restored by bronchoscopic surgery. Bronchoscopic reexamination showed only an ulcerous lesion with full bronchial lumen. Microscopic study revealed the rest of the tumor intramurally (see arrows). Further attempt at bronchoscopic surgery was inadvisable because of the danger of bronchial perforation. In both cases lesions in the distal parenchyma indicated external surgery (bilobectomy and pneumonectomy).

Naturally an adenoma must be radically extirpated but sacrifice of intact functional parenchyma is unnecessary (as is common in cases of carcinoma).

Many bronchologists believe that even the sessile type of bronchial adenomas (except cylindromas) are suitable for endoscopic extirpation and in cases of reported recurrence serial bronchoscopic surgery is justified. On the other hand some chest surgeons think that every bronchial adenoma should be treated with external surgery. Certainly only an entirely endobronchial adenoma is removable bronchoscopically. A sessile tumor may extend into the deeper layers of the bronchial wall (Figs 91, 92, and 93G and H) or into the parenchyma ("iceberg type"); therefore radical extirpation of the tumor cannot be proved (Figs 91 and 92). The author believes that indication for radical removal of an adenoma is certain only if it is pedunculated (Figs 87-89). If after seemingly radical extirpations an adenoma repeatedly recurs the "iceberg type" of adenoma must be considered as being present and the patient should be put in the hands of a surgeon. For removal of an adenoma bronchoscopy may be repeated two or three times but serial bronchoscopic treatment is indicated only in cases in which the patient is unsuitable for surgery (debilitated, too old) or if the patient refuses external surgery. In these cases in contrast to basal cell carcinomas the serial removal is not an error because it does not alter the histologic structure. Failures of bronchoscopic removal are demonstrated in Figures 91 and 92.

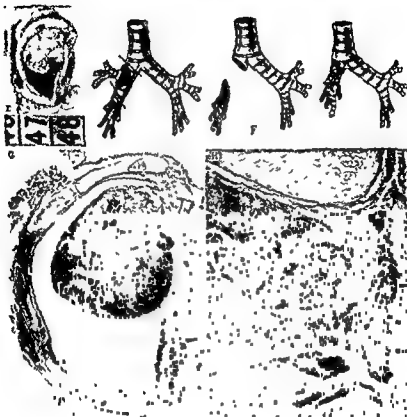


FIG 9. A Frontal view of the head showing the position of the tumor in the main and stem bronchi (see white arrow) but the extrabronchial shadow in connection with the intrabronchial one (see black arrow) suggest the "iceberg type" of adenoma. C Bronchogram showing the tumor as a filling defect (see arrow). Because of the suggestion of "iceberg type" of adenoma and the shrinking process of the upper lobe surgical treatment (lobectomy, bronchial resection and anastomosis) was chosen (see sketch F). Photograph of resected bronchial portion (E) shows the tumor to be totally intrabronchial but closer inspection of the tumor

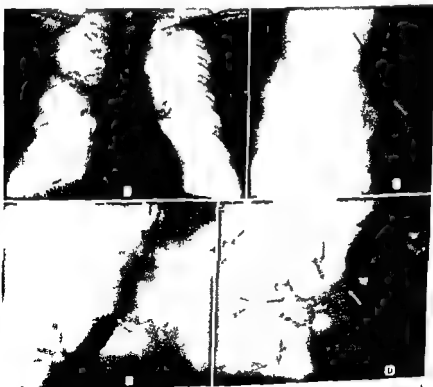
in the main and lower lobes 6 months after surgery the arrow points to the position of the anastomosis

adenocarcinoma and round cell carcinoma. Cells of the planocellular carcinoma are more differentiated than those of the other groups; cells of adenocarcinoma are less so; and cells of round cell carcinoma are the least differentiated. The stages of increasing malignancy of a tumor occur in this order:

1 The *planocellular carcinoma* grows relatively slower and less often causes metastasis to the lymph nodes than the others. It is inclined to necrosis and cavitation, extends peribronchially and diminishes the bronchial movements and narrows the bronchial lumen, sometimes having a craterous shape.

2 The *adenocarcinoma* appears chiefly in a lobar or segmental bronchus, frequently produces a circumscribed mass in the pulmonary parenchyma, often causes metastases in the lymph nodes, but rarely fixes the bronchial wall. Sometimes this tumor extends from the parenchyma into a bronchial lumen as a polypoid mass.

3 The majority of the *round cell carcinomas* arise from a main bronchus, infiltrate tissues rapidly, involve lymph nodes and mediastinal organs.



(See caption on facing page)

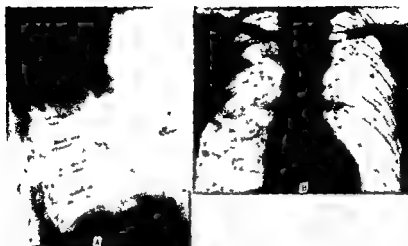


FIG 95 X rays showing atelectasis in the posterior segment of the right upper lobe due to bronchial carcinoma. A Anteroposterior B Lateral

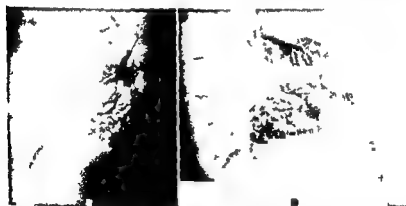


FIG 96 Aimed bronchograms show amputation (see arrows) of the two segmental bronchi of the right middle lobe by bronchial carcinoma which caused atelectasis in the middle lobe. A Anteroposterior B Lateral. The bronchograms were made in the standing position thus the contrast fluid filled the bronchi of the medial basal segment also. Tissue for histologic examination was obtained by blind biopsy. Pneumonectomy was performed.

in an early stage and readily fix and deform the bronchial walls. Metastases from the round-cell carcinoma to the bifurcational carinal lymph nodes widen the tracheal carina, extension of the tumor in this area may be so large as to obscure the original tumor in the bronchoscopic appearance.

The second system is the standard one of Broders,<sup>17</sup> in which the "histologic grading" is based on the percentage of the differentiated and undifferentiated cells in a tumor population. In Grade I undifferentiated cells are less than 25 per cent, in Grade II, between 25 and 50 per cent, in Grade III, between 50 and 75 per cent, and in Grade IV, above 75 per cent.

Examination of surgical specimens shows that tumor cells creep from the visible tumor in a proximal direction under the mucosa about 1.5 cm in planocellular carcinoma, approximately 2 cm in adenocarcinoma, and even further in round-cell carcinoma. It has also been proposed that a specimen be taken for biopsy from the seemingly intact mucosa, proximal to the visible tumor. All these facts must be considered when the question of operability is under discussion.

The *alveolar-cell carcinoma* is considered separately. This tumor is rare. It is located in the periphery and is also called *diffuse carcinoma* or *diffuse pulmonary adenomatosis*. Its diagnosis is not easy, bronchoscopically it is invisible. It may be recognized by cytologic examination.

Metastasis of a carcinoma from other organs into the lung may be by direct hematogenous spread, indirect hematogenous spread through the thoracic duct, and directly from contiguous organs. Biopsy or bronchial lavage in these cases is also important, because the location of the primary tumor (e.g., carcinoma of the thyroid gland, hypernephroma, chorion epithelioma) may be detected from the characteristic histologic appearance.



FIG. 94. X rays showing atelectasis in the anterior segment of the right upper lobe due to bronchial carcinoma. A. Lateral. B. Anteroposterior. C. Photograph showing the resected upper lobe with the tumor inside the bronchus.



FIG. 90 Chest x rays showing atelectasis of the left upper lobe of a sixty year old man with collapse to the anterior mediastinum A Lateral B Anteroposterior C Anteroposterior bronchogram shows normal bronchial tree at first impression but thorough examination completed by the lateral bronchogram (D) proves the upper lobe bronchus to be obstructed by the bronchial carcinoma. This case clearly demonstrates the need for orientation in three dimensions (See also Fig 90C and D )

**Signs and Symptoms** In the beginning *symptoms* of a bronchial carcinoma are very subtle. However, for early diagnosis these subtleties should be recognized. They are *cough*, alternating in character, *pain*, starting with slight discomfort in the chest cavity and increasing until at last it becomes very severe, *hemoptysis*, blood streaking, *microhemoptysis*. *Dyspnea* and *stridor* signalize pronounced stenosis and hoarseness suggests involvement of one of the recurrent laryngeal nerves.

The x ray appearance is extremely variable. As frequently mentioned in the foregoing paragraphs, carcinoma with stenosis or occlusion of a bronchus may cause atelectasis involving a lung, a lobe or a segment



FIG 97 Chest x rays showing atelectasis in the lateral basal segment of the right lower lobe due to bronchial carcinoma: A Lateral B Anteroposterior



FIG 98 X rays showing atelectasis in the posterior basal segment of the right lower lobe of a fifty year old male due to a carcinoma in the segmental bronchus A Lateral B Anteroposterior (From the collection of the Chevalier Jackson Clinic Philadelphia)

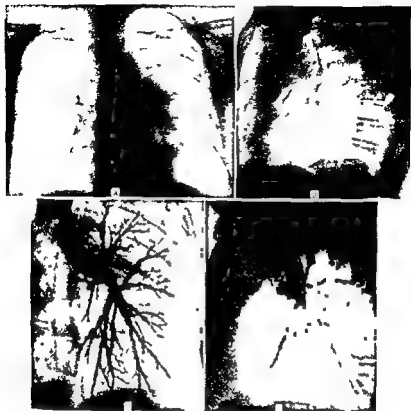


FIG. 99. Chest x rays showing atelectasis of the left upper lobe of a sixty year old man with collapse to the anterior mediastinum. A Lateral B Anteroposterior C Anteroposterior D Anteroposterior.

**Signs and Symptoms** In the beginning *symptoms* of a bronchial carcinoma are very subtle. However for early diagnosis these subtleties should be recognized. They are *cough*, alternating in character, *pain* starting with slight discomfort in the chest cavity and increasing until at last it becomes very severe, *hemoptysis*, blood streaking, *microhemoptysis*. Dyspnea and stridor signalize pronounced stenosis and hoarseness suggests involvement of one of the recurrent laryngeal nerves.

The x ray appearance is extremely variable. As frequently mentioned in the foregoing paragraphs carcinoma with stenosis or occlusion of a bronchus may cause atelectasis involving a lung, a lobe or a segment.





FIG 100 Chest x-rays of a fifty-six year-old man showing atelectasis in the posterior subsegment of the left upper lobe due to bronchial carcinoma. A Anteroposterior B Lateral

(Figs 94-102), and permanent or recurrent pulmonary inflammation which in the past have been mistaken for virus pneumonia. Abscesses arise from necrosis of the carcinomatous or pneumonic tissue. The x-ray shadows of all these changes are well known, but the shadows may be confluent with that of the tumor itself, and, furthermore, the picture may be varied also by sanguinous exudative pleuritis. The x-ray shadow of the border of the tumor is rather uneven, yet is rounded, in some cases, its margin radiates like a polyp. The x-ray appearance of a carcinoma usually is not characteristic.

**Diagnosis.** The examinations for diagnosis should be performed in the following order: (1) complete physical examination, (2) fluoroscopy, (3) laboratory examinations (the blood picture is generally normal, sedimentation rate accelerated, and anemia is secondary, in the sputum *Mycobacterium tuberculosis* and tumor cells should be searched for), (4) x-ray films, in every case at least anteroposterior and lateral, must be taken (Fig 96), and often oblique films or planigrams as well, (5) bronchoscopy (biopsy, aspiration, or lavage), (6) bronchograms, (7) angiocardio-gram, if necessary, (8) needle biopsy, and (9) exploratory thoracotomy. If the first seven types of examination fail to reveal a correct diagnosis, the last two should be done.

The treatment of a bronchial carcinoma at the present time is pneumonectomy and only in rare cases lobectomy. The results of surgical therapy are still disappointing. The most successful surgeons are able to demonstrate 5-year survival in 20 per cent of the operatively treated cases, but in only 1 per cent of the diagnosed cases. Only 4 to 17 per cent of the diagnosed cases are operable. The reason for these discouraging facts

s that the interval is too long between the first symptoms and the diagnosis. According to the best statistics the average duration of this period is almost a full year. 4 months pass before a patient first visits his physician. 1 1/2 months until the first x ray is made and then an average of 4 1/2 months before the correct diagnosis is established.

The difficulty in recognition of malignant bronchial tumors and the diagnostic errors are frequent because the initial symptoms of carcinoma are very similar to those of less serious diseases such as simple chronic bronchitis and the usual cough of smokers. Pulmonary carcinoma also imitates other more severe diseases than chronic bronchitis such as prolonged pneumonia, abscesses and pulmonary tuberculosis and therefore the diagnosis made from clinical and x ray examinations alone is very uncertain. The diagnosis can be proved only by exact morphologic examinations for which a specimen is obtainable by bronchoscopy, excision of an external lymph node, needle biopsy or exploratory thoracotomy. However, these examinations are easily neglected by both the patient and the physician. In metastasis to an external lymph node the patient has no hope of recovery, needle biopsy may seem dangerous to the patient and his physician and thoracotomy is a relatively major operation. Bronchoscopy reveals the most accurate result with the least inconvenience and hazard.

The bronchologist makes a diagnosis of bronchial carcinoma by direct and indirect signs. The *direct* sign is the bronchoscopically visible tumor. The *indirect* signs are stenosed, fixed or deformed bronchi, broadened or obliterated tracheal carina, etc.

The bronchologist can obtain specimens for morphologic examination by three methods: specimen for biopsy taken from the visible tumor, so-called



FIG. 101. Chest x rays showing atelectasis in the anterior segment of the left upper lobe due to bronchial carcinoma in a fifty-eight-year-old man. A. Anteroposterior. B. Lateral.



FIG. 102. Chest x rays showing atelectasis in the lingula due to bronchial cell carcinoma. The specimen was obtained for histologic examination by blind biopsy. A. Anteroposterior. B. Lateral.

blind biopsy and aspiration of bronchial secretions for cytologic examination.

**BIOPSY.** The specimen should be removed in a pea- or bean-sized fragment (or even smaller portions) in one piece. From a piece of tissue perhaps 1 mm in diameter the critical diagnosis of a malignant tumor and the determination of the degree of malignancy are often hardly attainable. Taking a biopsy specimen from a visible tumor is an almost harmless procedure; the resultant slight bleeding stops very soon.

**BLIND BIOPSY.** When bronchoscopy shows no lesion in the bronchus of an involved lobe or segment but from the presence of lobar or segmental atelectasis occlusion of the bronchus is indicated, the occluding tumor should be suspected in a deeper bronchoscopically invisible area of the supplying bronchus. In these cases forceps may be "blindly" inserted into the bronchus to obtain a specimen of the suspected tumor.<sup>16</sup> By this method an adequate specimen has often been obtained for histologic examination (Figs. 96 and 102). Obviously, blind biopsy must be a very gentle manipulation. The malignant tissue grasped by the forceps is easily detached; therefore this method of taking a biopsy specimen is generally a very easy procedure. The bronchologist knows the direction of the nearest bronchial spur and bites parallel to this spur. However, if the forceps grasp a bronchial spur and the bronchologist feels that he is pulling an elastic resistant tissue, the forceps are immediately opened and the tissue released. It has been learned that this procedure is entirely harmless.

**CYTOLOGIC EXAMINATION.** For cytologic examination secretion is aspirated if possible directly from the bronchus of the involved parenchyma. If an adequate amount of secretion is not obtainable, saline solution is injected into the bronchus and reaspirated. Secretion or clots in the solu-

tion are then measured on a glass slide fixed in an ether alcohol mixture and stained with Papanicolaou's stain tumor cells are then searched for in these preparations. By this same method tumor cells may also be found in the sputum. Material obtained either bronchoscopically or from sputum must be smeared and fixed within 3 hours or the tumor cells will be destroyed.

Bronchial carcinoma can be diagnosed by these methods in from 75 to 80 per cent of the cases. The most accurate results are obtained by histologic examination of a specimen obtained by biopsy. In the early diagnosis of the bronchoscopically invisible tumor cytologic examinations of bronchial secretion obtained by aspiration or lavage are very important. Every available method must be used to improve the persistently poor results in treatment of bronchial carcinomas.

### Summary

Bronchial tumors are divided into three groups: benign, borderline (adenoma) and malignant. Benign bronchial tumors are relatively rare. Their bronchoscopic removal is generally not difficult. Two types of bronchial adenomas are differentiated: the solid or carcinoid and the glandular or cylindroma. Adenomas occur in 5 per cent of all cases of bronchial tumors. Some authors consider adenomas benign, some acknowledge that they may undergo malignant degeneration and others believe that they are actually or potentially malignant. An adenoma grows endobronchially, intramurally or into the pulmonary parenchyma. The last type is called the "iceberg type" of adenoma.

In the very beginning symptoms are absent. Later the tumor causes simple bronchial stenosis followed by valvular emphysema, recurrent atelectasis and pneumonia from which in the last period abscess or bronchiectasis develops. Repeated profuse sometimes perilous hemoptysis is the most pronounced symptom. X-ray signs are not characteristic. Actually only bronchoscopy renders clinical recognition of an adenoma possible. Bronchoscopic biopsy is essential for accurate diagnosis.

Radical bronchoscopic removal of an adenoma is certain only if the tumor is pedunculated. If after seemingly radical extirpation an adenoma repeatedly recurs, if it is the intramural or iceberg type or if it has caused irreversible change in the distal parenchyma, external surgery is indicated.

The incidence of bronchoalveolar carcinoma has increased truly and markedly in the last two decades. Now it occurs as often as stomach malignancies and perhaps actually more frequently.

A pulmonary carcinoma can be located in a main or lobar bronchus (central or hilar), in the tertiary to quaternary bronchus (intermediate) or near the pleura (peripheral) or may be a diffuse carcinoma.

Histologically the bronchogenic carcinomas are divided into three groups: planocellular, adenocarcinoma, and round cell carcinoma or they are graded by the percentage of differentiated and undifferentiated cells in a tumor population.

Early symptoms are discomfort in the chest, cough, pain, and micro hemoptysis. Later dyspnea, stridor, hoarseness and pulmonary suppuration develop. X ray signs are extremely variable.

The following diagnostic examinations should be performed: complete physical, fluoroscopic, laboratory x ray, bronchoscopic, and if necessary bronchographic, angiocardigraphic, needle biopsy, and exploratory thoracotomy.

The bronchoscopist may observe direct or indirect signs of a carcinoma. He can obtain specimens for morphologic study by routine biopsy, blind biopsy, and aspiration of the bronchial secretion for cytologic examination.

The treatment of a bronchogenic carcinoma is pneumonectomy and only in rare cases lobectomy.

## Foreign Bodies

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Until the end of the last century bronchial foreign bodies were diagnosed if at all from data of the history and physical examinations. In chronic or uncertain cases they were recognized only if patients expelled them spontaneously but often they were revealed only by autopsies. Until the end of the seventeenth century therapy was frequently unsuccessful. Emetics were administered or attempts were made to shake out the foreign body by turning the patient upside down. At the end of the seventeenth and beginning of the eighteenth century tracheotomy and gentle irritation of the tracheal mucosa were performed in an attempt to have the patient expectorate the foreign body. According to statistics foreign bodies were expectorated spontaneously and without any treatment in 46 per cent of the cases with emetics and shaking in 3 per cent of the cases but about half of the patients died. After tracheotomy was instituted only 27 per cent died. Over all the mortality in tracheal foreign bodies was about 30 per cent and that in bronchial foreign bodies about 50 per cent.

Objects slipping into the pharynx from the mouth or sometimes from the nose are swallowed into the esophagus or stomach in the majority of cases. They are inhaled into the lower airways most frequently by the airflow of a forced inspiration. Fright, crying and laughing are accompanied by quick deep inspiration. (A dramatic actor expressing fright on the stage gesticulates wildly with his arms and breathes quickly and deeply.) This type of rapid strong airflow may carry a foreign body through the normally widened glottis into the trachea and bronchi. For example this can happen when a child or even an adult is rocking on his chair during a meal and suddenly falls backward or a child while holding a mouthful of food is quickly and unexpectedly hit in the face (Case 10) or an object slips suddenly from the mouth into the laryngopharynx of a playing child or a working adult (dressmaker, upholsterer). Such unexpected circumstances may cause fright and result in inspiration of an object. The following case serves as a characteristic though unusual example.



FIG 103 A Anteroposterior x ray showing atelectasis of the left lung due to a tack (B) in the left main bronchus of an eight year-old boy (see Case 8) Anteroposterior chest x rays were taken 11 days (C), and 3 months (D) after the removal of the foreign body and show progressive improvement

**CASE 11** X-ray films showed a tack in the main bronchus of an eight year old boy (Fig 103A) The father explained that the boy had been hit by a motorcycle 5 weeks before and received only some minor injuries but had been breathing heavily since this accident This explanation was believed to be an attempt on the part of the father to try to get more money from the motorist Only after a few days, when the foreign body was removed and the boy was reassured, did it become evident that the father had told the truth The boy explained what had happened Unknown to his father, he had found a bright copper tack on the road When the motorcycle had hit him, being frightened, he had put his "treasure" into his mouth and in fright aspirated it

As a rarity, it should be mentioned that a worm may climb from the intestines through the esophagus into the trachea and also that from outside the body a pocketknife blade, projectile, or a piece of shrapnel may enter the airways

A foreign body may be aspirated also during medical manipulation. For example, during tooth extraction, a piece of the crown may split, the dental canal reamer may roll out of the hand of the dentist (Fig. 104), a duodenal catheter may slide into the trachea and the metal end break off from a worn rubber tube by the shearing action of the spasmodically closed glottis (Fig. 105), during tonsillectomy or adenoidectomy a piece of tissue may fall into the trachea, or during bronchoscopy a blade of the forceps or the small electric bulb may break off (Fig. 106).

During sleep, narcosis, drunkenness, or an epileptic attack, unrecognized foreign objects may be aspirated (Fig. 59). Sometimes a foreign body inspiration may remain unrecognized even in the conscious state of a patient for a reason to be explained later.

Inspiration of a foreign body may occur in every age group but it is most frequent in childhood, the child may put nearly any small object into his mouth.

A foreign body may become impacted in the larynx remain in the trachea or slide into the bronchi.

In the larynx of an adult, an object larger than the laryngeal lumen



FIG. 104. Chest x rays showing a dental canal reamer in the left posterior basal segment of a forty-six year-old man. A. Anteroposterior. B. Lateral. After an unsuccessful bronchoscopic attempt it was coughed up in the larynx and its point penetrated the posterior pharyngeal wall from which it was easily removed (1). Another case was observed in which the dental reamer also changed its position. This was surprising because of the very sharp point of the dental canal reamer.







FIG. 107 A Anteroposterior chest x-ray was completely negative in the case of a one-year-old boy whose respiration had been dyspneic and stridorous for several months. His physician had diagnosed tracheal stenosis due to compression by a tuberculous lymph node (see Case 9). Direct laryngoscopy showed marked circular congestion and some gray elevation in the subglottal region. B Papilloma was diagnosed by histologic examination. C Repeated laryngoscopy revealed a foreign body, a springlike thin bone which was imbedded in the overlying mucosa causing congestion and papilloma-like hypertrophy of the epithelium misleading even the pathologist.

loma or cyst but it may also be a foreign body. The difficulty of diagnosis in certain cases is demonstrated by the following example.

**CASE III** The respiration of a one-year-old child had been dyspneic and stridorous for several months. The child was referred by his physician with the diagnosis of tracheal stenosis due to tuberculous lymph nodes. X-rays were completely negative (Fig. 107A). The stridor was most pronounced with stethoscope at the laryngeal level. Direct laryngoscopy showed marked circular congestion and some gray elevations in the subglottal area. Because of the possibility of papilloma the gray elevations were removed for histologic study. This proved the diagnosis of papilloma exactly (Fig. 107B). The condition of the patient was unaltered. With the aim of removing the remaining papilloma tissue laryngoscopy was again performed with general anesthesia but only smooth congested mucosa was observed and no papilloma. The examination might have been discontinued but then a very thin white line looking like scar tissue was detected. With rather routine moving of the forceps this little tissue was grasped and after the forceps were withdrawn very surprisingly a paper-thin bone 4 by 25 mm was seen between the blades (Fig. 107C). It was equally surprising to the parents who were unable to give any explanation for the presence of the foreign body.

The infant aspirated a bone at eight or nine months of age. During the next 3 or 4 months the springlike bone dug into the overlying mucosa and caused congestion and papilloma-like hypertrophy of the epithelium misleading even the pathologist. The precise (and it must be confessed, somewhat lucky) endoscopic search led to the accurate diagnosis and therapy.

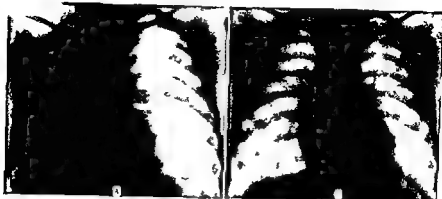


FIG 108 Anteroposterior chest x rays A An atelectasis (drowned lung) of the right lung of a two year old girl due to a bean in the right main bronchus B Normal condition after removal of the foreign body

After inquiry of the patient or relatives *external examination* of the chest should be performed. These examinations are very important particularly in infants and children. From indrawing of the suprasternal notch of the suprascavicular and intercostal spaces and epigastrium and from retarded respiratory motion on one side laryngeal tracheal or bronchial stenosis will be determined.

The physical signs may vary from case to case or at different times in the same patient. They depend upon the condition of the distal pulmonary parenchyma. Decreased vocal fremitus, impaired percussion note, diminished intensity or even absence of the breath sounds may be noted. The particular sound called *stridor* due to bronchial stenosis and thus to the foreign body itself was discussed in Chapter 2.

The next step is *fluoroscopy* of the chest organs by which an opaque foreign body may be seen and with rotation of the patient its position determined. Nonopaque foreign bodies may be localized with fluoroscopic observation of the respiratory dynamic mechanism (Chap. 2).

Roentgenograms should also be taken to determine the position and shape of opaque bodies. The location of a foreign body must be determined in space (in three dimensions) therefore at least two films, an anteroposterior and a lateral are required. Involvement of the distal parenchyma will be determined also by these films which may yield further information as to the location of the body (Figs 103-108-113 and 115). Metal bodies project dense shadows, bones and shelled seeds (plum cherry, etc.) project less density, and soft seeds or other soft bodies are radioparent.

Physicians have been misled in the past by foreign bodies in the trachea or one of the main bronchi. These foreign bodies were frequently thought

to be located in the esophagus and in some cases an unnecessary esophagotomy was even performed. These errors may be avoided by space orientation or by Holzknecht's sign.

The important means of investigating stridor were discussed in Chapter 2. Dyspnea and its various types were explained in Chapters 2 and 3 and x-ray symptoms of the pathologic respiratory mechanisms in Chapter 11. The significance of the Holzknecht sign was demonstrated by an example (Case 1). Case 1 also represents an example of the very worthwhile conclusions that may be drawn from correlation of the Holzknecht sign, stridor, and dyspnea. Severe dyspnea may be caused in the presence of stenosis of *one* main bronchus not only by stenosis of the larynx or trachea but also by stenosis of the *other* main bronchus. As an example the following case is cited:

**CASE 10.** A two-year-old boy was forced by his father to eat a walnut; the boy refused to swallow the masticated pieces, was hit on the face, and in fright aspirated the walnut. On clinical examination he was found to be extremely dyspneic with inspiratory indrawing noted in the neck and epigastrium. Respiratory sounds were inaudible on the left and over the lower portion of the right lung field. Roentgenograms (Fig. 113) showed a displacement of the upper portion of the mediastinum to the right and the entire left lung and lower two-thirds of the right lung were emphysematous. The upper lobe showed some density and the left diaphragm was depressed. Fluoroscopy showed that the displaced upper portion of the mediastinum shifted with every inspiration toward the left side.

Foreign bodies were present in the left main bronchus and in the right stem bronchus; there was valvular emphysema of the whole left lung and of the right middle and lower lobes.

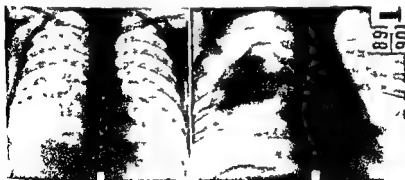


Fig. 113. a

b



FIG. 110 A B A machine screw in the right stem bronchus of a two year-old boy causing valvular emphysema in the lower lobe and collapse of the middle lobe D Lateral x ray showing no changes after removal of the foreign body (C)

Two pieces of walnut were removed one the size of a pea from the left main bronchus and another the size of a fairly large lentil from the right stem bronchus

The question is which considerations resulted in the exact diagnosis. Be

emphysematous and the left leaf of diaphragm descended therefore a lesion had to be located in the left main bronchus causing valvular emphysema. This was correct but the severe dyspnea was still unexplained somewhere in other stenosis had to be present and actually the right middle and lower lobes

were emphysematous to the same degree as the left lung thus the other foreign body had to be located in the right stem bronchus. This explanation was supported by the absence of respiratory sounds on the left and on the lower portion of the right. The child was breathing freely only with his healthy right upper lobe. The *Holzkecht* sign yielded the greatest help toward the accurate diagnosis.

The airway may be stenosed by a foreign body not only in the larynx, trachea, or bronchi, but also in the pharynx, primarily in the laryngopharynx, and may produce severe dyspnea. In the laryngopharynx, a large piece of meat will often impact, overlying the introitus of the larynx, and cause severe dyspnea or perhaps death by asphyxia. However, in the majority of cases it can be reached by the finger and easily removed.

The following case represents another example of the interesting investigations carried out by the bronchologist.

**CASE 11** An infant about one year old put into his mouth a part of a relatively small pacifier from which the ring and the rubber nipple were absent. This object slipped back into the pharynx and the child began to choke. The mother dipped her finger into the child's pharynx and touched the foreign body, which then disappeared. The infant was then intermittently dyspneic. The relatively large object certainly found no room in the larynx and trachea of the infant, and it was not found in the laryngopharynx. It might have slipped down into the esophagus compressing the lumen of the trachea now and then depending on the child's posture. With fluoroscopy no changes in



FIG. 111. Chest x-rays of atelectasis of the lateral basal segment of a seventy-two-year-old woman due to gold dental crown (C) in the corresponding bronchus. A. Lateral. B. Anteroposterior.



FIG 112 Chest x rays of a six year-old boy taken in (A) expiration and (B) inspiration showing valvular emphysema in the right lung due to a fragment of walnut in the right main bronchus



FIG 113 A Anteroposterior x ray of a boy aged two years shows valvular emphysema of the left lung and of the right middle and lower lobe due to fragments of walnut in the left main bronchus and in the right stem bronchus. The child was breathing freely only with his healthy right upper lobe. B X ray taken 5 days after removal of the foreign bodies showing a normal condition on both sides (Case 10)

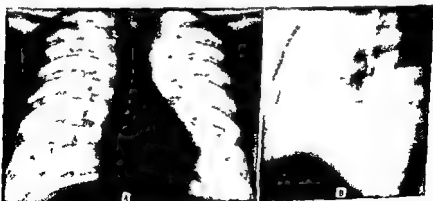


FIG 114 X ray films showing a pin in the left angular superior segment of a four year-old boy. A Anteroposterior B Lateral. The pin was removed bronchoscopically

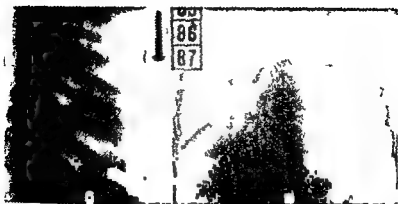


FIG. 115 X-ray films showing shadow of pneumonia in the right middle and lower lobe due to a screw in the sternum of a three-year-old boy. A. Later I. P. Anteroposterior C. Photograph of the removed foreign body 27 mm in length.

the chest organs were observed but curiously when his head was elevated by a hand on his jaw and his mouth shut he immediately began to suffocate. This observation showed that the foreign body was located in the nasopharynx where the mother's hand had pushed it. Before fluoroscopy nobody had noticed that the infant became dyspneic only when his mouth was shut. The foreign body was easily removed from the nasopharynx.

Hundreds and hundreds of additional examples could be cited but even with these it could not be emphasized too strongly how prudent physicians including bronchologists must be in searching for foreign bodies.

The circumstances of the removal of foreign bodies from the trachea or bronchi are generally simple and evident. The procedure is completely mechanical therefore the bodies are grouped by their shape—one, two and three dimensional and hollow bodies.

The most frequently occurring one-dimensional foreign bodies in the lower airway are pins (Fig. 114), nails (Fig. 103) and screws (Figs. 110 and 115). Two-dimensional ones are buttons, flat bones (Fig. 107) and coins. Three-dimensional ones are beans (Fig. 105), peas, peanuts, walnuts (Figs. 112 and 113), watermelon seeds, corn kernels, fruit stones (Fig. 61), pearls (Fig. 116), bones. The most frequently found hollow subjects are cartridges (Fig. 63).

Preliminary selection of forceps suited to the size and shape of the given foreign body should be made in every case. A test of forceps with a duplicate of the foreign body is a very important step in preparation for bronchoscopic manipulation. Without adequate forceps or other suitable instruments it is impossible to remove foreign bodies. In the majority of cases insufficient preparation or poor instrumentarium is the simple reason





FIG 116 X ray films showing a pearl in the right lower lobe bronchus of an eight year-old girl A Lateral B Anteroposterior C Photograph of the foreign body 1 cm in diameter Collapse of the lower lobe is clearly shown in the lateral film but it is hidden behind the heart in the anteroposterior film

for unsuccessful efforts or prolongation until the patient is exhausted. The bronchologist must know the precise circumstances of the insertion and the function of his instruments (forceps, hook, etc.) he must feel the depth at which the distal end of his instruments work, he must conceive and recognize the relation of the organs, bronchoscope, instruments, and the foreign body to one another from the bronchoscopic appearance. He also must learn what can be done with forceps, hooks, and other auxiliary instruments through the bronchoscope. No field of medical science exists where the principle of *nil nocere* is more important than in bronchial foreign bodies. Except in an emergency it is less harmful to leave a foreign body in the lung until the patient can be admitted to a well equipped suitable institution than to torture him and traumatize the air passages until he becomes very ill or moribund. It is extremely dangerous to enter the lower airways with any instrument until the examiner becomes familiar with the mechanical problems and precise technique of bronchoscopic foreign body extraction.

### Summary

Foreign bodies are inhaled into the lower airways most frequently by airflow of a forced inspiration which occurs in cough, fright, crying, or laughing. A small object also may be aspirated during sleep, narcosis, drunkenness, or epileptic attack, and also during medical manipulations.

Closely following the aspiration the patient coughs heavily. Later irritation from the foreign body subsides (latent period). This is the reason why many foreign bodies in the lower airway become chronic.

## FOREIGN BODIES

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Signs of an acute foreign body are caused by sudden mechanical irritations and stenosis or obstruction. In chronic cases inflammations prevail. In searching for a foreign body the patient's history is very important and if the slightest suspicion emerges the patient must be sent without delay to a suitable institution. The diagnosis of subglottal laryngitis or diphtheria must never be accepted without visualization of the larynx. In infants and young children direct laryngoscopy must be performed in every case of laryngeal stridor.

Inspection of the chest and physical signs are also important. Indrawing of the suprasternal notch and intercostal spaces and epigastrium diminished intensity or even absence of the breathing sounds or stridor may lead to the discovery of a foreign body. Fluoroscopic respiratory mechanism.

It must be emphasized that physicians including bronchologists must be extremely prudent in searching for foreign bodies which in their acute stage can cause unexpected sudden suffocation and if they are chronic certainly cause severe pulmonary suppuration. Negligence in any case may become a lethal mistake.



## Differential Diagnosis

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Symptoms signs and diagnostic methods can be briefly summarized as follows

**Patient's History** The rapid onset of symptoms (cough dyspnea stridor etc) indicates acute respiratory or pulmonary infection or a foreign body. Symptoms lasting several months or a few years indicate subacute or chronic laryngitis or bronchitis (especially sinobronchitis in children) abscess bronchiectasis pulmonary tuberculosis aneurysm new growth in the larynx bronchi or mediastinum or a foreign body in its latent stage. Long duration of symptoms for several years or even decades may be caused by chronic respiratory or pulmonary infections and a foreign body but long duration in connection with recurrent hemoptysis in a young person is very characteristic of bronchial adenoma.

The patient's history is most important in searching for a foreign body. Inquiry and spontaneous explanation by the patient or parents reveal very important data in acute cases of a foreign body or in cases of chronic pulmonary suppuration due to foreign body.

**Signs and Symptoms** Cough is the most common symptom of a respiratory or pulmonary disease. It can be dry or productive. Dry cough appears in cases of chronic laryngitis aneurysm or new growth in the larynx bronchi or mediastinum. Productive cough is present in cases of abscess bronchiectasis pulmonary tuberculosis tumor or foreign body. Odor of sputum may mean abscess bronchiectasis or foreign body.

A brassy bitonal sonorous cough is very characteristic of diseases with stenosis of the trachea or main bronchi due to external pressure or in fewer cases to endobronchial lesions. This type of cough usually appears in children because of pressure or bronchial rupture of a tuberculous endothoracic lymph node or pressure of esophageal foreign body and in adults because of compression by an aneurysm. The cough is usually spasmodic in cases of bronchial asthma tuberculous bronchitis and closely after the aspiration of a foreign body.

Cough frequently first becomes manifest after an intercurrent simple respiratory infection even in cases of chronic pulmonary diseases such as

epithelialized abscess, *bronchiectasis* pulmonary tuberculosis, or tumor

The time of maximal incidence of the cough is morning or night (at bedtime) in cases of nasal sinusitis, morning in cases of heavy smoking night in cases of abscess or bronchiectasis and in cases of bronchial asthma any time, but most often at night or on awakening from sleep

The cough is a very significant factor in spreading the mediastinal and traumatic interstitial emphysemata

Pain is characteristic in none of the diseases discussed in this book. Pulmonary diseases may cause only slight discomfort in the chest but also an increasingly severe pain. Severe pain is almost always present in cases of trauma of the 'lung tree'. In connection with lung diseases the pain is generally visceral or deep in the chest, which causes the patient to breathe faster and to cough

Fever, increased sedimentation rate, leukocytosis, malaise, and other symptoms which are very common in acute pulmonary infections (acute bronchitis, pneumonia, abscess, etc.) occur also in chronic diseases with acute exacerbations (chronic and deforming bronchitis, bronchiectasis, and tuberculosis). Usually the fever is irregular or intermittent

*Hemoptysis* is one of the most alarming symptoms of a bronchopulmonary disease and frequently a challenging problem to a bronchologist. Bronchial, pulmonary, cardiovascular, and blood diseases may cause hemoptysis. True hemoptysis may occur in almost every bronchopulmonary disease. Among bronchial diseases, it occurs most frequently in acute bronchitis, acute exacerbations of chronic bronchitis, bronchiectasis, bronchogenic carcinoma, bronchial adenoma, and foreign body, among pulmonary diseases, in tuberculosis, cysts, parasitic diseases, trauma of the 'lung tree'.

The color of the blood is usually, bright red, and at least a part of it is frothy. The blood may be pure or mixed with sputum. After a severe attack the sputum is almost always bloodstained for a few days, usually dark in color, and clotted. If the blood is swallowed the stools are tarry.

The hemoptysis is frequently profuse, and pure blood is coughed up in cases of dry bronchiectasis, abscess, advanced tuberculosis, and adenoma.

The sputum may be bloodstained or blood streaked in cases of wet bronchiectasis, pulmonary tuberculosis or mycosis, bronchitis, abscess, malignant tumor, and in a latent stage of a foreign body.

A small amount of pure blood might be coughed up in any cases of the above diseases, and also in cases of cysts and in acute crises of foreign bodies.

*Stridor* and *tracheal stenosis*. The sound differs according to the

a wheezing or whistling sound, a loosely fixed foreign body

lated tumor may cause a sound similar to a rattle or the roll of a drum.

Inspection of the chest is very important in every case of suspected endothoracic disease. *Inspiratory indrawing of the supraclavicular or sternal epigastric and intercostal soft tissues* indicates laryngeal tracheal stenosis or a stenosis on both sides in major bronchi. During quiet respiration stenosis or even occlusion of a main bronchus does not necessarily cause indrawing of these tissues especially in children but during exercise or when a child cries these signs become pronounced. The intercostal spaces are wider than normal in cases of valvular emphysema and narrower in cases of atelectasis or other kinds of shrinking processes on the affected side or the affected portion of the chest. In both cases the affected side or portion of the chest is hindered in respiratory movements.

*Dilated veins of the frontal wall of the chest* indicate stenosis of the vena cava superior due to compression of a lesion in the mediastinum (caval sign).

*Dyspnea* may be of the reflex or physiochemical type.

In normal conditions the Hering Breuer reflex limits both inspiration and expiration. If normal pressure in the chest cavity rapidly changes afferent impulses signalize pathologic condition which may upset respiratory balance even in cases of healthy cardiorespiratory organs. This phenomenon frequently occurs in tracheotomized children during decannulation or in other conditions when the Hering Breuer reflex becomes hypersensitive by reduced distensibility of the lungs resulting e. g. from edema, congestion, inflammation, fibrosis or tumor. This abnormal sensitivity inhibits the inspiratory phase earlier and causes a more rapid shallow type of breathing.

*Clubbing fingers* are seen characteristically in morbus caeruleus and also in association with fibroid lungs or in almost every disease that leads to persistent congestion of terminal parts of the fingers (emphysema, chronic bronchitis, pulmonary abscess, bronchiectasis, lung tumor, etc.).

**Diagnostic Methods.** *Fluoroscopy* is an important method in discovering and locating a pulmonary lesion or a foreign body. In cases of bronchial stenosis the whole process of respiration must be observed. Attention must be focused chiefly on the motion of the roentgen middle shadow and of the diaphragm. It frequently happens that one can diagnose a foreign body or other bronchial stenosis only by directing his attention to the Holz knecht sign. Differentiation between valvular emphysema and atelectasis may often be intricate without this sign.

In bronchography, fluoroscopic control of the optimal filling of the bronchi is a *sine qua non* for a correctly taken bronchogram. In timed bronchography, the accurate placing of the special catheter and filling of the bronchi also must be controlled by fluoroscopy.

A ray appearance of an atelectasis, lobar or segmental pneumonia and

an abscess is, generally, characteristic. In cases of atelectasis, the shadow shows the pulmonary unit diminished in size, which indicates bronch obstruction. A typical lobar or segmental pneumonia is shown by a mass shadow on the pulmonary unit which is normal in size and shape (Figs 50, and 51). Two other kinds of pneumonia may appear in this category: the atelectatic and the abscess form.<sup>26</sup>

The shadow of an atelectatic form of pneumonia is smaller than a typical one and shows the pulmonary unit diminished in size. If a lobe is fixed by pleural adhesions the interlobar margins of the shadow appear concave in shape<sup>27</sup> (Figs 44, 49, 63, and 81). This type of shadow also indicates bronchial obstruction.

An abscess type of pneumonia projects a shadow larger than the pulmonary unit. The lobe or segment is extended by secretions, and its margins are convex in shape (Figs 37 and 46).

X-ray findings are very characteristic in cases of an open abscess, showing a shadow similar to a basket with a handle. The body of the basket represents the fluid and the handle the wall above the air of the cavity (Figs 53 and 54).

In cases of chronic bronchitis, bronchiectasis, pulmonary tuberculosis, mycosis, and bronchogenic tumors, the x-ray findings are extremely variable. X-ray films of a patient with chronic bronchitis may show some marked bronchial branchings or may show normal conditions; bronchiectasis projects shadows of several cavities and shrinkage of one or more pulmonary units; sometimes no pathologic evidence may be shown on x-rays. X-ray findings of pulmonary tuberculosis, mycosis, or tumor are so complex that they cannot be discussed briefly. The roentgenographic shadow diagnostic procedure is very helpful in most cases; however, it seldom reveals the accurate diagnosis. An opaque foreign body can be diagnosed by x-ray, but in many other conditions, even if the shadows are as characteristic as those of atelectasis, pulmonary abscess, or pneumonia of a pulmonary unit, the x-ray diagnosis is uncertain and inadequate.

The next step toward the accurate diagnosis is bronchoscopy. The bronchologist diagnoses a disease from *direct and indirect bronchoscopic signs*.

The direct signs are what the examiner observes in the lesion itself. In cases of pathologic conditions in bronchi accessible to bronchoscopic exploration, the bronchologist may diagnose the condition correctly at first sight. However, even in these most accurate cases, further studies (histologic, bacteriologic, etc.) may be necessary.

If a lesion is located out of the vision of the examiner (outside the bronchial lumen or in the periphery), indirect signs can be appraised. Abscess or bronchiectatic cavities are never seen through a bronchoscope.

Peripheral inflammations tumors and foreign bodies are hidden behind the pulmonary parenchyma but the indirect signs—secretions blood distortion stenosis rigidity of the visible bronchi—exhibit their presence and location. In these cases specimens are available by bronchoscopic aspiration for bacteriologic study by lavage for cytologic examinations and by blind biopsy for histologic study.

In still uncertain cases *bronchography* may assist. Bronchograms show deformed bronchi abscesses bronchiectasis cysts or a benign tumor by filling defects (Figs 82 and 93) or accordion like deformity by an arial compression they show stenosis or truncated bronchi in cases of malignant tumors (Fig 96). In cases of filling defect in a pulmonary unit aimed bronchography by which surprising results may be obtained is indicated (Figs 55 56 59 62 67 79 and 80).

*Tests of pulmonary function* may reveal data for early detection of pulmonary dysfunction in patients considered to be healthy on the basis of clinical and radiologic examinations. In a known disease a specific diagnosis may be made with pulmonary function tests which could not be established by other methods.

For completion of the diagnostic methods the needle biopsy angiograms and exploratory thoracotomy should be mentioned. In modern diagnosis the exploratory thoracotomy occupies the same place which was held by the laparotomy some decades ago.

Today all these methods must be included in the armamentarium of modern diagnosis and therapy.





## Selected References

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- 1 AERY C. *Der Bronchialbaum der Säugetiere und des Menschen*. W. Englemann Leipzig 1880
- 2 ALBRECHT W. "Die Fremdkörper der Trachea und der Bronchien" in A. Denker and O. Kihler (eds.) *Handbuch der Hals-Nasen-Ohrenheilkunde*. Springer Verlag Berlin and J. F. Bergmann Munich 1929 vol III pp 918-954
- 3 AUGUSTIN V. and L. BJÖRDER. Segmentale Röntgenschatten bei Pertussis. *Monatsschr Kinderh* 103:409 1955
- 4 BALÓ J. *Lungenkarzinom und Lungenadenom*. Verlag der Ungarischen Akademie der Wissenschaften Budapest 1937
- 5 BALÓ J., D. KÁNYAI and A. BIKFALVI. Die Bronchiolduktoome und ihre Bedeutung. *Acta morphol* 5:71 1953
- 6 BECKER H. Das Beatmungsbronchoskop nach Friedel. *Deut. Gesundh. wirtsch* 11:221 1936
- 7 BENEDICT E. B. *Endoscopy*. The Williams & Wilkins Company Baltimore 1951
- 8 BIRNBAUM G. L. *Anatomy of the Bronchovascular System: Its Applications to Surgery*. Year Book Publishers Inc. Chicago 1954
- 9 BOJDER E. A. A critique of the international nomenclature on bronchopulmonary segments. *Dis. Chest* 23:266 1953
- 10 BOYDER T. A. *Segmental Anatomy of the Lung*. McGraw Hill Book Company Inc. New York 1955
- 11 BROCK H. C. *The Anatomy of Bronchial Tree*. Oxford University Press New York 1947
- 12 BRODERS A. C. Microscopic grading of cancer. *S. Clin. North America* 21:947 1941
- 13 BRUN M. and P. G. GERLINGS. Bronchoscopy in pulmonary atelectasis in children. *Acta oto laryng* 24:222 1935
- 14 BRUNING W. *Die direkte Laryngoskopie, Bronchoskopie und Oropharyngoskopie*. J. F. Bergmann Wiesbaden 1910
- 15 COMBES J. H., H. E. FOSTER, A. B. DEBOIS, W. A. BRISCOE and F. CALSEN. *The Lung: Clinical Physiology and Pulmonary Function Tests*. Year Book Publishers Inc. Chicago 1957
- 16 CURTIS J. K., H. K. RASMUSSEN and E. CRIST. Clinical evaluation of pulmonary function tests. *W. Clin. North America* 43(1):17 1959
- 17 DAHLER T. B. and L. F. HAUZE. *Diseases of the Chest*. The Williams & Wilkins Company Baltimore 1952

- 18 DENECKE, H J *Die oto rhino-laryngologischen Operationen*, Springer Verlag, Berlin, 1953
- 19 DUBOIS DE MONTREYNAUD, J F, R J EDWARDS, and A J GLADU A new method of bronchoscopy with cinematography and still photography, *J Biol Photographic A*, 24 45, 1956
- 20 FOURESTIER, M, A GLADU, and J VULMIERE Perfectionnements a l'endoscopie médicale Realisation bronchoscopique, *Presse méd*, 60-1292, 1952
- 21 FROSTE, N Bronchoscopy in pulmonary tuberculosis, *Acta tuberc scandinav* (Suppl), 23, 1950
- 22 GOLDMAN, A Malignant nature of bronchial adenoma of the bronchus *J Thoracic Surg*, 18 137, 1949
- 23 GORGÉNYI GÖTTSCHE, O *Tuberkulose im Kindesalter*, Springer-Verlag Vienna 1951
- 24 GORGÉNYI GÖTTSCHE, O, and D KASSAY Importance of bronchial rupture in tuberculosis of endothoracic lymph nodes, *AMA Am J Dis Child*, 74 166 1947
- 25 GORGÉNYI GÖTTSCHE, O, and D KASSAY Zur Bedeutung der bronchial Perforation bei der Tuberkulose der endothoracalen Lymphknoten *Schweiz med Wchnschr*, 80 1213, 1950
- 26 GORGÉNYI GÖTTSCHE O, and D KASSAY Atelektase im Kindesalter, *Ergebn ges Tuberk Forsch*, 14 391, 1958
- 27 GRAHAM, E A, T H BURFORD, and J H MAYER Middle lobe syndrome, *Postgrad Med*, 4 29 1948
- 28 HASLINGER, F *Directoskop Acta oto laryng*, 7, 1925
- 29 HAYEK, H *Die menschliche Lunge*, Springer Verlag Berlin 1953
- 30 HERNHEIMER H J *Bronchial Asthma*, Butterworth & Co (Publishers) Ltd, London 1952
- 31 HOFFMANN, F A "Emphysema und Atelektase," in H Nothnagel (ed) *Spezielle Pathologie und Therapie*, Alfred Holder, Vienna, 1900, vol XIV, part 2, p 1
- 32 HOLINGER, P H, and A H ANDREWS Bronchial obstruction, *Am J Surg*, 54 193, 1941
- 33 HOLINGER, P H "Endoscopic Photography and Otolaryngology and Broncho esophagology," in C and C L Jackson (eds) *Diseases of the Nose Throat and Ear*, W B Saunders Company, Philadelphia, 1945
- 34 HOLZKNECHT  
und Methodi
- 35 HORANYI, J
- 36 HUIZINGA E Eine internationale Regelung der Nomenclatur der Bronchi, *Pract oto rhino-laryng* 12 109 1050
- 37 HUIZINGA, E La Bronchostenose, *Les Bronches*, 1:71, 1951
- 38 HUIZINGA, E, and G J SMIET *Bronchography* Van Gorcum & Co, Assen, Netherlands 1949
- 39 JACOBÆUS, H C, P FRENCHNER and S BJARRMAN Some attempts at determining volume and function of each lung separately, *Acta med scandinav*, 71 174, 1932
- 40 JACKSON, C Overlooked foreign bodies in the air and food passages *Brit M J*, 11 686, 1925

- 41 JACKSON C Bronchial obstruction *Dis Chest* 17:125 1950
- 42 JACKSON C and C L JACKSON *Bronchoscopy Esophagoscopy and Gastroscopy* W B Saunders Company Philadelphia, 1934
- 43 JACKSON C and C L JACKSON *Diseases of the Air and Food Passages of Foreign Body Origin* W B Saunders Company Philadelphia 1936
- 44 JACKSON C and C L JACKSON *Bronchoscopy* W B Saunders Company Philadelphia 1950
- 45 JACKSON C and C L JACKSON *Diseases of the Nose Throat and Ear* W B Saunders Company Philadelphia 1959
- 46 JACKSON C L Anesthesia for peroral endoscopy *Ann Otol Rhin & Laryng* 49 1043 1940
- 47 JACKSON C L Indications and technic of bronchoscopy *J Internat Coll Surgeons* 435 1943
- 48 JACKSON C L New instrumentarium for peroral endoscopy and endoscopic photography *Laryngoscope* 67 777 1957
- 49 JACKSON C L and I F HUBER Correlated applied anatomy of bronchial tree and lungs with a system of nomenclature *Dis Chest* 11 319 1943
- 50 JACKSON C L and A R JUDD Role of bronchoscopy in treatment of pulmonary abscess *J Thoracic Surg* 10 179 1940
- 51 JACKSON C L and F W KOZELLEMAN So-called adenoma of the bronchus *Ann Otol Rhin & Laryng* 50 1264 1941
- 52 JACKSON C L and C M NOHUS The role of bronchoscopy in diagnosis and treatment of bronchial adenoma *Dis Chest* 20 353 1951
- 53 KALLAY I A gyűrűs tüdőscoposzkóp (Bronchoscope with ring mirror) *Orv Hetil* 99 1615 1959
- 54 KASSAY D Von den Symptomen der Bronchialstenose *Monatsschr f Ohrenheilk* 81 225 1947
- 55 KASSAY D The bronchial tree A classification and nomenclature *Acta oto laryng* 37 355 1949
- 56 KASSAY D A Tüdő Segmentumai (Segments of the Lung) Akadémiai Kiadó Budapest 1950
- 57 KASSAY D Bronhoszkóp és csecsemő és gyermekkorban (Bronchoscopy in infancy and childhood) *Cyermékgyógyzat (Pediatrics)* 1 113 1950
- 58 KASSAY D Zur Frage der Nomenclature der Bronchi *Monatsschr f Ohrenheilk* 85 55 1951
- 59 KASSAY D A new bronchoesophagoscope with proximal illumination *Acta oto-laryng* 42 64 1952
- 60 KASSAY D Pathologic respiratory mechanisms *Acta med Acad sc hung* 3 91 1952
- 61 KASSAY D Remarks on the international nomenclature of bronchopulmonary segments *Dis Chest* 20 610 1951
- 62 KASSAY D Hol Knecht's Sign kongressjelentés von der Tagung der Österreichischen oto-laryngologischen Gesellschaft und der internationalen broncho-oesophagologischen Gesellschaft Vienna 1956 p 115
- 63 KASSAY D Szélepcs-Legzési Mechanizmusok (Valvular respiratory mechanisms) *Tud Orr-és Gyógygyaztat (Otorhinolaryngology)* 2 2 1956

- 64 KASSAY, D. *Middle Lobe Syndrome*, William B. Dorman, Collingdale, Penn., 1957, p. 35
- 65 KASSAY, D. Observations on one hundred cases of bronchial foreign body, *AMA Arch Otolaryng*, 71 42, 1960
- 66 KASSAY, D., A. BIAFALVI, and J. BALÓ. Bronchialadenome, *Thorax chirurgie*, 3 24, 1955
- 67 KASSAY, D., D. DIMITROV SZOKODI, and G. MIHÓK. Die endoskopische Behandlung der Bronchialfisteln, *Thoraxchirurgie*, 3 250, 1955
- 68 KASSAY, D., M. ERDELYI, and R. SCHUSTER. Celzott Bronchographia (Aimed bronchography), *Radiol hung*, 7 78, 1955
- 69 KASSAY, D., K. GERGELY, and M. PÁNYI. Koraszülöttek Tüdőtelektáziájának Korszerű Gyógyítása (Up to-date treatment of atelectasis of premature infants) *Gyermekgyógyászat (Pediatrics)*, 1953
- 70 KASSAY, D., and Z. LABAS. Studium des Bronchiallumens bei Neugeborenen mit Entwicklungsstörungen, *Monatsschr f. Ohrenheilk*, 91 52, 1957
- 71 KASSAY, D., and Z. LABAS. Study on the intersegmental septa, *Dis. Chest*, 35 625, 1959
- 72 KEENEY, E. L. *Practical Medical Mycology*, Charles C. Thomas, Publisher, Springfield, Ill., 1955
- 73 KENDREY, G., J. JUMASZ, and J. BALÓ. Bronchiektasiák Kísérletes Előidézése (Experimental bronchiectases), *Kísérlet Orvostud*, 293, 1955
- 74 KOVÁTS, F., JR. and Z. ZSEBOR. Röntgenanatomische Grundlagen der Lungenuntersuchungen, Akadémiai Kiadó, Budapest, 1955
- 75 LABAS, Z. Chronikus Bronchitis. *Fül Orr Gégyógyászat (Otorhinolaryngology)*, 1 74, 1955
- 76 LABAS, Z., and G. MIHÓK. Bronchoszkopos "vak" probakimetszés (Bronchoscopic "blind biopsy"), *Fül Orr Gégyógyászat (Otorhinolaryngology)*, 1 74, 1955
- 77 LEMOINE, J. M. L'Ulceration bronchique tuberculeuse, *Ann. oto laryng*, 62 291, 1944-1945
- 78 LEMOINE, J. M., and A. DIACOMMOPOULOS. La separation des expectorations. *Rev. tuberc*, 8 117, 1943
- 79 LEMOINE, J. M., A. DIACOMMOPOULOS, and J. PAILLAS. La Bronchoaspiration des cavernes pulmonaires tuberculeuses ballonnées, *Rev. tuberc*, 10 205, 1944-1945
- 80 LOSCHKE, H., and K. ROKSTANSKY, quoted by C. Hirt and E. Mayer. Kehlkopf, Lufttröhre und Bronchien, in F. Henke and O. Lubarsch (eds.) *Handbuch der Speziellen Pathologischen Anatomie und Histologie*, Springer-Verlag, Berlin, 1928, vol. III, part 1, p. 500
- 81 MARKÓ, D. Zur Diagnostik der pleuralen Adhäsionen, *Fortschr. Röntgenstr*, 41 451, 1930
- 82 MILLER, W. S. *The Lung*, Charles C. Thomas, Publisher, Springfield, Ill., 1950
- 83 MOERSCH, H., and O. T. CLAGETT. Pulmonary cysts. *J. Thoracic Surg*, 16 179, 1947
- 84 NORRIS, C. M. Foreign bodies in the air and food passages. A series of two hundred fifty cases, *Ann. Otol. Rhin. & Laryng*, 57 1019, 1948

- 85 NORRIS C M J LONG M J OPPENHEIMER and M R WESTER  
Bronchography Apparatus and technique *J Thoracic Surg* 17 357 1948
- 86 OVERHOLT R H and L LANGER A new technique for pulmonary segmental resection Its application in treatment of bronchiectasis *Surg Gynec & Obst* 84 257 1947
- 87 OVERHOLT R H and L LANGER *The Technique of Pulmonary Resection* Charles C Thomas Publisher Springfield Ill 1949
- 88 Panel Discussion Anesthesia in bronchoesophagology *Ann Otol Rhin & Laryng* 64 598 1955
- 89 POPPE T K Comfortable bronchoscopies using the sitting position *Tr Am Bronchoesophagological A* 33 153 1958
- 90 PROETZ A W *Applied Physiology of the Nose* Annals Publishing Company St Louis 1953
- 91 RHODIN J Ultrastructure of the tracheal ciliated mucosa in rat and man *Tr Am Bronchoesophagological A* 39 89 1959
- 92 DI RIZZO S *The Bronchus* Charles C Thomas Springfield Ill 1949
- 93 ROGGEN G T DENIAUX and A DUPREZ Stenose bronchique experimentale chez le chien *Acta clin belg* 7 445 1952
- 94 ROCKEY E E Surgical technic of tracheal fenestration *J Thoracic Surg* 96 486 1957
- 95 RUBIN H T and B M L KULLY Topical anesthesia in bronchoscopy *A M A Arch Otolaryng* 56 13 1952
- 96 SPILL M S *Severe Bronchial Asthma* Charles C Thomas Publisher Springfield Ill 1950
- 97 STEINBERG T H L *Therapy of Fungous Diseases* Little Brown & Company Boston 1955
- 98 STEINMANN C P Die Patho-Physiologie des Bronchialbaumes *Fortschr der Hals Nasen Ohrenheilk Separatum* vol 3 1956
- 99 STORDY S E Cystic disease of the lung *J Thoracic Surg* 18 404 1949
- 100 STUTZ E Bronchographische Beiträge *Fortschr Röntgenstr* 72 129 1949
- 101 VEGHLYI P V T T KEMÉNY and J Sós Bronchial changes in experimentally induced cystic degeneration of pancreas *A M A Am J Dis Child* 79 846 1950
- 102 WARFAMBOURG H and P GRAY *Pathologie des zones pulmonaires* Masson et Cie Paris 1947



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